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ABSTRACT

Since childhood lead poisoning first gained recognition as an important public health problem, the concept of lead poisoning has been examined and revised repeatedly. This national conference was convened to review and examine the current state of the problem, prevention activities, and recent studies on the toxic effects of lead at very low levels. Papers presented at the conference focused on the following topics: (1) changing sources of lead poisoning; (2) national perspectives; (3) overview of low-lead toxicity; (4) low-level exposure and children's development; (5) U.S. Department of Housing and Urban Development regulations; (6) the Environmental Protection Agency's perspective on lead in water, gasoline, and soil; (7) regional collaboration; (8) laboratory issues; (9) environmental issues; (10) legal issues; (11) lead used as treatment in folk medicine and other nonpaint sources of lead; and (12) future directions. (JD)

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CHILDHOOD LEAD POISONING

CURRENT PERSPECTIVES

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HYATT REGENCY
INDIANAPOLIS, IN
DECEMBER 1, 2, 3 1987

PROCEEDINGS OF THE NATIONAL CONFERENCE, CHILDHOOD LEAD POISONING: CURRENT PERSPECTIVES

December 1, 2, 3, 1987

This conference was made possible by support from:

U.S. Department of Health and Human Services, Bureau of Maternal and Child Health and Resource Development.

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Harvey I. Sloane, M.D., Jefferson County Judge/Executive, Jefferson County Fiscal Court and Mason Rudd Chairman, Louisville-Jefferson County Board of Health.

Indiana State Board of Health, Bureau of Family Health Services, Division of Maternal and Child Health.

University of Indianapolis, Center for Continuing Education for Nurses.

PREFACE

The last decade has seen a change in varying degrees of lead toxicity. The more we learn about lead exposure, the more apparent it becomes that even a little may be too much. Lower level lead toxicity has become a national health problem of major concern. The Childhood Lead Poisoning: Current Perspectives Conference re-emphasized the challenge of reducing childhood lead poisoning.

The conference provided an opportunity to:

- identify the changing sources of lead poisoning;
- express awareness of the current trends in the national lead poisoning problem;
- recognize the long term effects of low level lead toxicity;
- express awareness of recent developments in the problem of low level lead;
- summarize the Department of Housing and Urban Development (DHUD) regulation as related to childhood lead poisoning;
- recognize the Environmental Protection Agency's (EPA) perspective of hazards of lead in water, gasoline and soil;
- discuss the significance of the March 1987 American Academy of Pediatrics statement on lead poisoning;
- describe future directions in childhood lead poisoning management;
- describe the significance of integrating lead screening as a [illegible] child health service;
- understand the significance of laboratory issues as related to standardization of EP extraction methods, oxidizing reagents and new instrumentation;
- recognize the issues related to environmental investigation methods and hazard abatement;
- increase awareness of the legal processes available for the implementation of statewide and local legislation;
- discuss medical management strategies;
- recognize the specific effects of lead on pregnancy;
- recognize the risk of lead poisoning posed by folk medicines, pottery and other non-paint sources.

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WORKSHOP PROGRAM

A. *Hud Regulations*

Moderator	Mildred Fort, M.H.S.A. Lead Program Coordinator Illinois Department of Health
Speaker	Carolyn Newton Office of Public / Indian Housing Department of Housing and Urban Development

B. *Regional Collaboration: NECCLPP* (Wed.)

Moderator	Linda McGee, R.N. Child Health Nurse
Speakers	Amy Zimmerman, M.P.H. Coordinator, New England Consortium of Childhood Lead Poisoning Programs Division of Family Health Rhode Island Department of Health
	Martha M. Turner, R.N., B.S.N. Coordinator, New Hampshire Childhood Lead Poisoning Prevention Program
	Phyllis Madigan, B.A. Chief of Laboratory, Massachusetts Childhood Lead Poisoning

C. *Laboratory Issues*

Moderator	James Simpson, M.P.A. Public Health Advisor Centers for Disease Control
Speakers	Patrick J. Parsons, Ph.D. Section Head, Lead Poisoning Program Laboratory of Inorganic and Nuclear Chemistry; Assistant Professor, Department of Environmental Health and Toxicology, School of Public Health Sciences, SUNY, Albany
	Noel Stanton, M.S. Chemist, Toxicology Laboratory State Laboratory of Hygiene, Wisconsin

D. *Environmental Issues*

Moderator	Raymond Tyler, R.S. Regional Environmental Health Consultant Maternal and Child Health/Public Health Services
Speakers	Lawrence Chadzynski, R.S., M.P.H. Chief, Center for Environmental Health Sciences Michigan Department of Public Health
	J. Julian Chisolm, Jr., M.D. Francis Scott Key Medical Center

E. *Legal Issues*

Moderator	J. Michael Meyer, III, R.S., B.A. Lead Program Coordinator Louisville-Jefferson County Board of Health
Speaker	Ed Schoenbaechler, J.D. Goldberg and Simpson, P.S.C.

F. *Medical Management*

Moderator	Dwala Griffin, R.N. Administrator of Preventive Medicine Louisville-Jefferson County Board of Health
Speakers	J. Routt Riegart, M.D. Associate Professor, Department of Pediatrics Medical University of South Carolina
	John W. Graef, M.D. Director, Lead Toxicology Clinic Children's Hospital Boston

G. *Effects of Lead on Pregnancy*

Moderator	Jane Lin-Fu, M.D. Pediatric Consultant Division of Maternal and Child Health
Speaker	David Bellinger, Ph.D. Neuroepidemiology Unit, Children's Hospital

H. *Folk Medicine and Other Non-Paint Sources of Lead*

Moderator	Carl Henn, M.S.P.H. Lead Program Manager Marion County Health Department
Speakers	Alan Ackerman, Ph.D. Research Assistant Professor Department of Family and Community Medicine
	Roberta D. Baer, Ph.D. Department of Anthropology University of South Florida

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INTRODUCTION

Jane S. Lin-Fu, M.D.

The prevention of lead poisoning among U.S. children is one of the most significant successes in recent public health programs. From a disease that was highly prevalent but little known only 20 years ago, lead poisoning has gained wide recognition as an important preventable cause of childhood mortality and morbidity. Since passage of the 1971 Lead-Based Paint Poisoning Prevention Act, much has been accomplished, but much more remains to be done. This national conference was convened to review and examine the current state of the problem, prevention activities under the Maternal and Child Health Block Grant, and recent studies on the toxic effects of lead at very low levels. It is hoped that the conference will bring into focus changes in public health policies and programs that are needed in order to respond to our current knowledge about the health effects of lead.

During the last two decades, both the scene on childhood lead poisoning in the United States and the very concept of lead poisoning itself have undergone dramatic changes. In the late 1960's and early 1970's, when mass screening of children first began, lead poisoning associated with prominent clinical signs and symptoms was common; even lead encephalopathy was not rare. But in the last 15 years or so, clinical lead poisoning has declined markedly as screening programs have expanded and identified children earlier and earlier. Today, the vast majority of children uncovered by screening programs have no overt clinical manifestation of the disease, and lead encephalopathy is encountered infrequently. This change is remarkable particularly because relatively little has been done to resolve the problem of lead paint on dwellings—an important source of lead poisoning in children. The success may be attributed to two major efforts: intense public education and mass screening of children, originally as a categorical grant program administered by the Centers for Disease Control, and since FY 82, as activities under the Maternal and Child Health Block Grant. Other contributing factors include the phased reduction of lead in gasoline and the decline in lead content of canned goods both of which have reduced the background exposure to lead.

Since childhood lead poisoning first gained recognition as an important public health problem, the concept of lead poisoning has been examined and revised repeatedly. It has evolved from a clinical syndrome that is generally associated with blood lead levels of 60 ug/dl or more to a condition manifested as subtle psychoneurological deficits and compromise in perinatal outcome, early growth and development that is associated with blood lead levels less than 15 ug/dl. It was only

in 1970 that the Surgeon General's Statement on the Medical Aspects of Childhood Lead Poisoning defined a blood lead level of 40 ug/dl or more as evidence of undue lead absorption. By 1985, the CDC Statement on Preventing Lead Poisoning in Young Children has lowered the limit for elevated blood lead level to 25 ug/dl. Many studies have now questioned whether this limit should be further lowered, and if there is in fact a threshold for lead injury in the very young.

The Bureau of Maternal and Child Health and Resources Development, Health Resources and Services Administration is pleased to support this conference and make the proceedings available to health workers who are concerned with the prevention of childhood lead poisoning. Special appreciation goes to Ms. Sarah Wilding, the Louisville, Kentucky Lead Poisoning Prevention Training Workshop staff, Ms. Naomi Johnson, Chairperson of the Conference Planning Committee and members of the Committee for their dedication and hard work which made this conference possible.

CONFERENCE PROGRAM

December 1, 1987

8:00 a.m.-12 Noon	Opening Plenary Session Hyatt Regency, Indianapolis, Indiana
8:00 a.m. Exhibits Open Moderator:	Naomi Johnson, R.D., M.S., Chairperson Indiana State Board of Health
8:30 a.m. Welcome & Opening Remarks	Denise E. Ingram, M.D., M.P.H. Bureau of Family Health Services Indiana State Board of Health
9:00 a.m. *Keynote Address: Changing Sources of Lead Poisoning*	J. Julian Chisolm, Jr., M.D. Francis Scott Key Medical Center
9:30 a.m. *The National Perspective*	Jane Lin-Fu, M.D. U.S. Department Human Services Division of Maternal and Child Health Bureau of Resource Development
10:00 a.m. *Overview of Low Lead Toxicity*	Herbert Needleman, M.D. Department of Psychiatry Children's Hospital of Pittsburgh
10:30 a.m.	Break
10:45 a.m. *Low Lead Exposure & Children's Development*	David Bellinger, Ph.D. Neuroepidemiology Unit, Children's Hospital
11:15 a.m. *Low Lead Level Toxicity*	Joel Schwartz, Ph.D. Department of Biostatistics Harvard School of Public Health
12:00 Noon	Luncheon
1:30 p.m.	Workshops, Session 1
3:00 p.m.	Break
3:30 p.m.	Workshops, Session 2

December 2, 1987

8:30 a.m. Exhibits Open	
Moderator	Mildred Fort, M.H.S.A. Illinois Department of Health
9:00 a.m. ***HUD Regulations***	Carolyn Newton Office of Public/Indian Housing Department of Housing & Urban Development
9:45 a.m. ***Lead in Water, Gasoline & Soil: EPA Perspective***	Ronnie Levin, M.A. U.S. Environmental Protection Agency
10:30 a.m.	Break
11:00 a.m. ***American Academy of Pediatrics, March 1987 Statement on Lead Poisoning***	John W. Graef, M.D. Director, Lead Toxicology Clinic Children's Hospital Boston
12:00 Noon	Luncheon
1:30 p.m.	Workshops, Session 3
3:00 p.m.	Break
3:30 p.m.	Workshops, Session 4

December 3, 1987

8:30 a.m. Exhibits Open	
Moderator	Sarah Wilding, R.N., B.S. Specialized Pediatrics Cabinet for Human Resources, Commonwealth of Kentucky
8:30 a.m. Workshop Summaries	Workshop Speakers
10:30 a.m.	Break
10:45 a.m. Future Directions	J. Routt Riegart, M.D. Associate Professor, Department of Pediatrics Medical University of South Carolina
11:45 a.m. Closing Remarks	Sarah Wilding, R.N., B.S. Specialized Pediatrics Cabinet for Human Resources, Commonwealth of Kentucky

WELCOME & OPENING REMARKS

Denise E. Ingram, M.D., M.P.H.

First of all, we are glad to have many of you who are visitors to Indianapolis to see our wonderful winter weather. Isn't it nice for the holiday season?

This conference is so timely and definitely necessary with the problem we face in the country with lead poisoning.

It is particularly important since the detrimental effects it has on our children are increasing and we are even hearing more about the fact that it has effects before birth and you will have very noted speakers and experts on this area who will be discussing that today. I am also very much concerned about the unequal effect lead has with its social economic ratio and geographics disproportion and you will be addressing that issue too. Many have called it the newest social disease and it is, in fact, that but I also call it intentional injury. Intentional adults to children, we know what causes it, it is totally preventable. We know all the issues involved but for some reason we haven't eliminated those.

You are here to address that. I am very pleased. It is very interesting that Naomi mentioned my experience with lead poisoning and my introduction to it. I was a first year medical student when I did the program in Rocksberry. Many of you know about Rocksberry. It is one of the poorest areas, or I should say low income areas, in Boston. I have mixed feelings about that experience. One being that it was encouraging when many of the pediatricians who coordinated and ran that program out of Boston City Hospital were just so committed to working on it and I was just a young bushy-eyed medical student and that encouraged me. It was also very frustrating because many of the authorities, those people who had control over various issues, weren't really interested in addressing it or it wasn't really high on their list of priorities.

I now have mixed feelings because ten years later we are addressing some of the same issues. I am very optimistic, I am optimistic because this year, well really fiscal year 1987, Congress appropriated additional funding to be targeted for child health programs. We at the Indiana State Board of Health have elected to expand and augment our lead poisoning prevention program. Now we have additional monies to make sure that we can cover areas in the state more widely and give the necessary funding for screening that is necessary and also to make sure that very important follow-up components are in place.

We are very pleased that Congress is now looking at this and other problems and setting priorities for us.

In addition, in the area of secondary prevention many of you may be aware of public law 99-457, which has as one of its major components early identification of infants and children at risk for developmental delay and other problems. We know that lead poisoning significantly has a component that leads to developmental delay, so we at the Board of Health are working with the Department of Mental Health and also with the Department of Education to make sure those children who have developed mental delays as a result of lead poisoning will be identified and get into treatment programs and other necessary secondary programs. We are very optimistic in addition to other federal agencies such as HUD which you will hear from later in this conference, that have taken a more pro-active and preventive stand on eliminating lead paint from housing.

So things have changed in ten years. It has been very interesting for me because being a physician the idea of time and emergency is much different from public health in that often an emergency means that if you don't act in two or three seconds you may have a dead child or a very seriously impaired child. Public health takes a little bit longer. It has taken a while for me to appreciate that time frame and I am still learning. I haven't settled totally for it yet but I just want to welcome you and I'm sure you share my optimism in addressing this problem and wish you a very successful conference. Thank you.

KEYNOTE ADDRESS

CHANGING SOURCES OF LEAD POISONING

J. Julian Chisolm, Jr., M.D.

Tetraethyl lead was discovered in the United States by Thomas Midgely in 1921, added to gasoline in 1923 and raised a public health controversy from the very beginning.[1] After intense public scrutiny during the past fifteen years, lead additives in gasoline are being phased out, so that after sixty-six years this most recently developed lead product is about to depart the scene. The use of lead solder in food cans is also being rapidly phased down under the auspices of the U.S. Food and Drug Administration; indeed, cans with lead soldered seams have been completely removed from all prepared infant food and juice containers. These two maneuvers by themselves have substantially reduced the baseline exposure of the general population to lead within the past decade. Thus, we have now come full circle back to the ancient sources of lead and lead poisoning in the United States today. Lead is one of the ancient metals. A lead statue in the British Museum, discovered in Turkey, dates from at least 3000 B.C. or almost 6000 years ago.[2] Lead has been mined, smelted and used in cosmetics, internal and topical medicinal preparations, paint pigments and glazes since earliest recorded history.[3] Among these ancient uses of lead, lead-based paints on residential housing constitutes the major public health hazard due to lead in the United States today. If we are to move toward primary prevention of lead toxicity in infants and young children, new and far more effective methods of identification and abatement of lead paint hazards will be needed. This will almost certainly involve new technologies. Likewise, new and better therapeutic modalities are urgently needed and may become a reality within the next several years.

During the 1970's, automotive exhaust from the combustion of leaded gasoline accounted for 90% of airborne lead. Concurrent with the phasedown in the lead content of gasoline and the reduced consumption of leaded gasoline, air lead levels declined substantially between 1978 and 1984.[4] During this period, median air lead concentrations declined from 1.2 to 0.4 micrograms/m^3, while the 90th percentile levels decreased from 3.2 to 0.6 micrograms/m^3. Between 1978 and 1980, the average blood lead concentration in the United States population declined from 15.9 to 9.6 micrograms/dl of whole blood. With the continued phasedown in leaded gasoline since 1984, there has

been a further reduction in airborne lead even in major cities, so that it is estimated that the average blood lead concentrations in the general U.S. population today may be about 5-6 micrograms/dl of whole blood. This decline in blood lead levels is attributable, not only to the substantial reduction in the consumption of leaded gasoline, but also to the decrease in food lead. I shall not discuss water lead as this topic will be covered later in this conference.

Since ancient times, lead has been used in cosmetics, particularly white lead, red lead and black lead. This practice continues today in the Old World. *Al kohl* or *surma*, as it is called in the Indian subcontinent, is used as a black eyeliner and to produce cosmetic marks on the faces of infant girls as well as adult women. Samples of this cosmetic have been shown to contain 70-85% of lead by weight. Even today, *al kohl* is used in topical medicinal preparations in the Middle East, including the treatment of the umbilical stump in newly born infants.[6] In the United States, imported internal and external folk remedies containing lead have been found among recent immigrants from Asia. Azercon and greta contain lead tetraoxide and are used medicinally among Mexican-Americans.[6] Lead is still used in the glazing of pottery and hardly a year goes by that we do not learn of a contaminated lot being removed from the market in the United States. The wide variety of sources of exposure of children to lead may be found in the CDC's 1985 Statement on Lead Poisoning[7] as well as in the American Academy of Pediatrics statement,[8] about which you will hear later.

I turn now to lead pigments. In ancient times lead pigments were used primarily by artists—a practice which continues today—and for the decoration of public buildings.[3] In England, the first recorded use of lead in house paint occurred in 1274 A.D.[2] The Dutch process, which greatly increased production, was introduced in the 17th century. Indeed, the first case of lead poisoning in a white lead worker was recorded in England in 1678. Among the 1217 lead workers with lead colic studied by Tanquerel des Planches in the 1830's, 70[7] were exposed to lead paint pigments either as house painters or in the manufacture of these pigments.[9] The manufacture of lead pigments, including white lead, continued in the United States until very recent years. Indeed, white lead was the almost universal white pigment used in house paints in the United States until about 1940, after which it gradually was replaced by titanium dioxide. It is now estimated that, perhaps, 40-50[7] of the currently occupied U.S. housing stock contains lead-based paints on exposed residential surfaces.

Serial blood lead measurements taken at three month intervals, from birth to thirty months of age, in the Cincinnati prospective lead study[10] show the clear relationship between blood lead level and the type of housing in which infants reside. All infants show a slight increase in blood lead between birth and six months of age. Thereafter, infants residing in modern public housing without lead paint hazards show stable geometric mean blood lead levels of about 12

micrograms/dl of whole blood. By contrast, infants residing in dilapidated pre-World War II housing show a steady increase between six and eighteen months of age during which geometric mean blood lead concentration increases to 25 micrograms/dl where it stabilizes at least until thirty months of age. In other words, one half of the children residing in old poorly maintained pre-World War II housing have unacceptable elevations in blood lead concentration.

For the past fifteen years, the management of childhood lead toxicity has been based on screening to identify the child with an elevated blood lead concentration, referral for medical evaluation and, in certain cases, chelation therapy. This approach has its limitations. To have any effect at all, it must be coupled with rapid identification and abatement of lead hazards in the child's environment.

An important principle of chelation therapy, as stated by Aaseth,[11] is as follows:

> "Enhanced excretion induced by a drug is meaningless from a therapeutic point of view if it is not paralleled by a decrease of the metal concentration in the critical organ."

In the case of lead, recent human and experimental studies indicate that the brain is the critical organ in the fetus and infant. With these points in mind, new and important experimental data on the use of calcium disodium ethylenediamine tetraacetate ($CaNa_2$ EDTA) raise serious questions, particularly in regard to the $CaNa_2$ EDTA Mobilization Test. Cory-Slechta et al have just reported a detailed study on the mobilization and redistribution of lead during the course of $CaNa_2$ EDTA therapy.[12] These workers chronically poisoned rats at a low level, producing pre-treatment blood lead concentrations of 25-40 ug Pb/dl of whole blood. Although the dosages of $CaNa_2$ EDTA used are not directly transferable from rats to humans, the dosages used were considered comparable to those used in children. Following a single dose of $CaNa_2$ EDTA, blood lead concentration decreased and urinary lead output increased sharply, as observed in humans. She also measured the changes in the concentrations of lead after a single dose of $CaNa_2$ EDTA in bone, kidney, liver and brain. Her data showed that the greatest decrease occurred in bone lead and that some decrease in kidney lead also occurred. The studies of others have indicated that approximately 85% of the lead mobilized by $CaNa_2$ EDTA is derived from bone. By contrast, a modest increase in the lead content of liver and a marked increase in the lead content of brain occurred indicating substantial redistribution of the lead during the mobilization. In the various experiments, brain lead was increased anywhere from 33-100%. After five days, there was no net loss of lead from either brain or liver, despite the fact that blood lead levels declined and that there was a marked increase in urinary lead output. Other recent studies have also shown, as observed in children, that the excretion of delta-aminolevulinic acid, a marker of lead's inhibitory effect on heme

synthesis, also decreases during chelation therapy. These data, as well as other considerations, raise serious concern about the use of $CaNa_2$ EDTA Mobilization Test in routine clinical practice.[13] Indeed, the animal data call for a re-evaluation of the use of chelating agents in the management of lead toxicity.

New drugs are needed and, perhaps, are on the horizon. Perhaps the most promising drug is 2,3-dimercaptosuccinic acid (DMSA), which in experimental animals has been shown to reduce the concentrations of lead in various soft tissues, including the brain. However, DMSA appears to have little effect in the rat on the lead content of bone. DMSA has also been shown to cause a sharp decrease in blood lead concentration and a marked enhancement of urinary output in lead poisoned adults. As with $CaNA_2$ EDTA, internal redistribution occurs following therapy even without further exposure, so that the soft tissue concentrations of lead after therapy rise toward the pretreatment levels. I know of only one experimental study in which DMSA and $CaNa_2$ EDTA have been administered chronically to the rat.[14] In this study, both drugs, when administered intermittantly over a six week period, reduced the concentration of lead in the brain significantly, but not to control levels.

Between 1978 and 1984, I studied a group of 184 children admitted to the hospital with blood lead $\geq$ 50 ug/dl for long-term chelation therapy.[15] They were then followed as outpatients for up to 2½ years thereafter. Among the 184 children, only 20 could be relocated to either modern public housing in good condition or totally gutted and renovated houses. Only in these 20, was there sustained improvement. Among the remaining 164 children who were returned to old houses abated in the traditional manner, 75 or 46% had a total of 127 recurrences of blood lead levels in excess of 50 ug/dl. These data clearly reveal still further the limitations in chelation therapy and call for a primary preventive approach based on reduction in exposure.

Traditionally, lead-based paints have been removed from woodwork primarily by burning, sanding and scraping. In Baltimore—and I suspect in many other cities—no professional cleanup of the debris has been required. A common practice has been to give an unemployed man a propane torch with which to burn and soften the paint, so that it could be scraped off down to the bare wood. Such workers generally wear no protection and are totally unsupervised. Such an approach fills the house with fine lead-bearing particulates, some of which have been shown to fall within the respirable range. How many "torch men" develop acute lead poisoning, I do not know. However, poisoning in workers removing lead paint in this manner is well known.[16,17]

In 1984 we had an opportunity in Baltimore to compare this traditional approach, with deleading carried out by city crews according to the principles outlined in the 1985 CDC Statement.[7] These city crews removed lead-based paint with heat guns and scrapers, cleaned extensively with ordinary vacuum cleaners and high phosphate detergents

TABLE 1

Summary of Household Dust Lead (PbD) Values (mcg/sq ft) over Time by Surface and by Abatement Group

Surface	Pre-Abate GM	Post-Abate GM	6 Mos. Post GM
Floors			
Traditional	234	1,282	307
City Crew	400	687	339
	p <.01	p <.01	ns
Window Sills			
Traditional	1,212	3,444	1,644
City Crew	2,284	907	1,936
	p <.01	p <.001	ns
Window Wells			
Traditional	12,016	12,570	12,815
City Crew	21,313	9,740	26,013
	ns	ns	p <.05

GM=geometric mean p values for t tests

and repainted the deleaded areas. This work was carried out by Mark Farfel, then a doctoral student at the Johns Hopkins University School of Public Health and Hygiene in collaboration with Evan Charney and myself.[18] The deleading process was monitored by obtaining household dust lead samples by the wipe technique from floors, windowsills and window wells. Table 1 shows geometric mean dust lead values as micrograms per square foot pre-treatment, post-abatement and six months later. There were 53 homes in the traditional group and 18 homes in the city crew group. Studies by others have shown that the floors in modern suburban homes and public housing units without lead hazards show mean values of 20 ug of lead per square foot with an upper limit of approximately 150 ug of lead per square foot. Pre-abatement, all surfaces were grossly contaminated and the degree of contamination increases if one moves from floors to windowsills to window wells. In the case of floors and windowsills, the abatement procedures significantly increase dust lead levels in the traditional group. The traditional wisdom has held that a child must bite on and chew discrete chips of paint. Since window wells do not present a biting surface, they were not treated by either group and remained a very rich source of lead-bearing particulates relatively unchanged even after six months. The window wells, indeed, often contained visible particles of paint. Perhaps, the most discouraging finding was the observation that no significant reduction in lead dust levels occurred even after six months.

In the vast majority of cases, blood lead concentrations in the children clearly increased in relation to the abatement. Indeed, among the 27 children residing in the homes abated in the traditional manner, 48% showed an increase in blood lead concentration greater than 5 ug/dl, including nine who were hospitalized for chelation therapy as a result of the abatement. Two, or 10.5%, of the nineteen children in the city crew group showed an increase in blood lead concentration to greater than 50 ug Pb/dl of whole blood and were hospitalized for chelation therapy.

Clearly, past procedures have been based on faulty concepts in that they have not taken into account the importance of particulate lead. That is not to say that children, particularly those with higher levels of lead absorption, do not ingest flakes of paint. Even so, the data of Bornschein et al,[19] indicate that lead in both paint and surface soil contribute to the lead content of household dust which in turn is significantly related to hand lead level, which in turn is related to blood lead level. Their data, in agreement with others, indicate that the major route of lead into the body of children with low level lead toxicity is via the hand to mouth route. Other studies have indicated that the lead in surface soil, particularly that adjacent to houses, is also derived from paint.

The considerations provide the scientific rationale for a new approach. Although experimental data show that dietary deficiencies of calcium, iron, zinc and excesses of dietary fat increase lead absorption, other factors may be equally if not more important. In human adult volunteers lead is far better absorbed when ingested in the fasting state than it is when administered with food.[20] Lead in street dust, while relatively insoluble of neutral pH, is highly solubilized and present in ionic form in 1.5 normal hydrochloric acid.[21] This may be taken as a model for the effect of gastric juice in solubilizing the lead salts in the dust. With regard to dusts, particle size may be even more important. In experiments in rats, Barltrop and Meek[22] fed identical quantities of lead to rats, varying only the particle size between 6 and 180 microns in diameter. Their data clearly showed an inverse relationship between absorption and retention of lead and particle size. The concentration of lead in the kidney was approximately seven times greater when lead was ingested in the smallest particles than it was when lead was ingested in particles of 180 microns in diameter. Particles <100 microns in diameter were the most efficiently absorbed. Que Hee et al[23] studied the distribution of lead according to particle size in household dust. Approximately 80% of the lead was found in particles less than 150 microns in diameter. Indeed, 21% of the lead was found in particles less than 44 microns in diameter. Such small particles cannot be trapped efficiently by the standard household vacuum cleaner. These data indicate that a high efficiency particle accumulator vacuum or HEPA vacuum is required for adequate decontamination. Indeed, all of our experience to date indicates that an effective approach to the

reduction of lead paint hazards in old housing must be similar to the approach used to reduce hazards due to asbestos.

During the past two years, we have undertaken experimental studies on lead abatement in vacant old houses in Baltimore.[24] In many of the old houses in which children with lead poisoning live, the floors are often splintered, pitted or have gaps between the floor boards, making it virtually impossible for the housewife to reduce the dust lead levels effectively through the ordinary means of cleaning. We have observed that dust lead levels are much lower on floors covered with vinyl or smooth linoleum. The effect of floor treatments on residual dust lead levels was studied in three vacant houses in which wooden floors in some of the rooms were treated, while those in other rooms were not. Table 2 shows the results. The target value for floors

TABLE 2

Effect of Floor Treatments on Residual Dust Lead Levels
Baltimore—1986

Group (N)	Residual Dust Lead (PhD) on Floors	
	<150 mcg/ft^2	≥ 150 mcg/ft^2
Treated† (22)	19	3
Untreated‡ (13)	2	11
		$X^2=14.32$
		$p<0.001$

†Treated=polyurethane, deck enamel then scrub and HEPA
Pbd: median, 78; range 24-360 mcg Pb/ft^2
‡Untreated=scrub and HEPA only
PbD: median, 480; range 84-1620 mcg Pb/ft^2

post-abatement has been less than 150 micrograms/ft.^{2} Clearly, treatment of wooden floors with polyurethane or deck enamel results in significantly better results than simply scrubbing the floors and vacuuming them with a HEPA vacuum.

In our more recent studies, we have been using replacement windows, off-site dipping of woodwork that can be easily removed and caustic stripping of woodwork not easily removed from the dwelling. Walls have been treated by encapsulation. If flooring is not in satisfactory condition, new flooring covered with vinyl or linoleum is put down. Table 3 shows the results in a dwelling in which these techniques have been used. This dwelling was unoccupied at the time of abatement, but was occupied immediately after the cleanup. Since we are interested in determining whether the occupant could maintain low dust lead levels, samples were taken two months following occupancy. Comparison of pre-abatement with three months post-abatement dust lead levels on floors and window sills show substantial improvement. However, window wells still remain a problem. Studies in this and

TABLE 3

House Dust Lead Levels Before and After Abatement
Experimental Dwelling No. 3

Site (N)*	Pre Abate	Post Abate	Post Paint & Floor Tx**	Post Cleanup***	3 Months Post
	10/31/86	5/19/87	6/19/87	6/27/87	8/26/87
FLOORS					
Tx**=coated (13)	522	857	31	473	188 (12)*
tiled (2)	1,017	1,220	605	255	77
WINDOW SILLS (6)	4,577	—	554	616	298
WINDOW WELLS					
replaced with vinyl unit (3)	26,398	2,740	3,234	1,287	1,891
not replaced (2)	34,858	—	10,111	8,368	10,129

Arithmetic Mean (micrograms/sq ft)
*(N)=(number of samples)
**Tx=treatment
***Cleanup: Single cleaning with high phosphate detergent followed by HEPA vacuuming

other experimental houses strongly suggest that replacement of the entire window unit may in the long run be necessary. In general, "deleading" and repainting of old wooden window units has not been satisfactory.

These studies, most of which have been carried out during the past three years in collaboration with the City of Baltimore, have led to the promulgation of new regulations for lead paint abatement by the City of Baltimore on 1 July 1987. The regulations apply to all interior surfaces and up to 4 feet on exterior surfaces, although all exterior woodwork about windows, doors and porches may be included in the near future. The salient features of these regulations are as follows:

1. Remove pregnant women, all young children until work completed.
2. Repair water leaks (roof, walls, plumbing).
3. Remove or seal all furnishings in plastic (including wall-to-wall carpet).
4. Removal of paint from woodwork: heat guns, replacement offsite or on-site stripping; (*NOT* permitted: torches, sanders); windows—entire unit including sill, sash, sashguides and wells.
5. Walls: repaint (minor defects) or cover with fiber glass, sheet rock, etc. and seal edges tightly.
6. Floors: cover with polyurethane, deck enamel, vinyl, linoleum.

7. Clean-up: HEPA vacuum, wet wash, HEPA vacuum, then *measure residual dust levels.*

The adequacy of abatement and cleanup will be keyed to achieving the following dust lead levels: Floors < 200 mg/ft.^{2}, windowsills < 500 mg/ft.^{2} and window wells < 800 mg/ft.^{2}

In summary, with the removal of lead additives from gasoline and the reduction of lead contamination of food, we have come full circle back to the ancient sources of lead and lead poisoning—those that have been with man for at least 5000-6000 years. There is little doubt that the major remaining public health problem with regard to lead exposure of children is the millions of tons of lead-based paints on the old housing stock. There are limitations to chelation therapy. It is doubtful that anyone has ever been "cured." At best, it is of little value unless the sources of lead in the child's environment are identified and effectively abated or removed. It is time to move toward primary prevention. This will require new approaches and new technology for the identification and abatement of lead paint hazards in housing. Abatement can no longer be left to the unsupervised torch man; rather, training and certification in the newer techniques will be needed. Proper disposal of wastes must be followed rather than dumping of the debris in the backyard or down the storm drain or sending it to the incinerator. Since particulate lead is a most important aspect of the hazard, the approaches that have been applied to asbestos, including the use of HEPA vacuums, will almost certainly be required for adequate cleanup after lead paint removal and/or encapsulation. Far more emphasis in the future must be placed on primary preventive approaches.

Finally, it has been 83 years since J. Lockhart Gibson first recognized the importance of particulate lead and hand-to-mouth activity in young children when he wrote as follows:

> "I . . . advance a very strong plea for painted walls and railings as the source of lead, and for the biting of fingernails or sucking of fingers, as . . . the means of conveyance of the lead to the patient." From J.L. Gibson, Australasian Med. Gazette (1904) *23*:149-153.

This in turn leads me to cite a quotation from Benjamin Franklin over 200 years ago:

> "The opinion of this mischievous effect from lead, is at least above sixty years old; and you will observe . . . how long a useful truth may be known . . . before it is generally receiv'd and practis'd on." Letter from B. Franklin to B. Vaughan, Philadelphia, July 31, 1786.

Let us hope that the rate of progress will now accelerate. Thank you.

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CHILDHOOD LEAD POISONING IN THE UNITED STATES: A NATIONAL PERSPECTIVE

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Introduction

Lead is an extremely useful metal that has found wide applications in both ancient cultures and modern civilization. In 1986, the world lead consumption totalled 5.6 million metric tons with one fifth of it in the United States. The major U.S. lead products include storage batteries, solder, ammunitions, cable covering, sheets, pipes and traps for buildings, lead oxides in paint, glass, and ceramics, and gasoline additive.[1] Through its diverse applications, lead has contributed immensely to our comfort and convenience, but for this we have paid a heavy price—the widespread pollution of our environment with a toxic non-biodegradable element. This in turn has created a man-made disease—lead poisoning. Today, childhood lead poisoning is one of the most common preventable pediatric problems in the United States where it results not only from modern applications, but also from age-old uses of folk remedies and cosmetics containing lead that are brought here by immigrants and refugees.[2]

The common occurrence of lead poisoning among U.S. children was first observed by Ruddock in 1924,[3] but the problem has attracted national attention only in the last 15-20 years. Congressional hearings in the late 1960's led to the passage of the 1971 Lead-Based Paint Poisoning Prevention Act. Mass screening of children and other prevention activities provided under the Act began in mid 1971 (FY 72).[4] Until the late 1960's and even into the early 1970's, many children had clinical evidence of lead poisoning; and lead encephalopathy was not an uncommon occurrence in the pediatric wards of many hospitals. But since mass screening and intense public education began, overt lead poisoning has become less common and lead encephalopathy rare. Today, the great majority of children discovered by screening programs to have elevated blood lead levels are clinically "asymptomatic." They appear well and would not have been identified through routine physical examination or laboratory tests such as complete blood count or urinalysis.

Most health care providers who have begun their careers in the last 10 years or so have never seen children with clinical illnesses resulting from lead poisoning. They have not cared for victims of lead poisoning who were left with severe mental retardation or paralysis. However, for those who have worked in the field somewhat longer, the memory of children entering hospitals with protracted vomiting, con-

vulsions, or coma from lead poisoning is still very vivid. Having seen such tragedies, watching the dramatic decrease in clinical lead poisoning in recent years has been an extremely exciting, gratifying, and encouraging experience. The achievements of the past decade have demonstrated that childhood lead poisoning, once thought by many to be too complex a problem to have a solution, is preventable.

But these dramatic achievements have produced a paradoxical effect. As clinical lead poisoning cases diminish, some health care providers have questioned if childhood lead poisoning is really still a problem? The answer is yes-yes-yes! Lead poisoning in children is a tenacious problem that cannot be eradicated easily.

Fifteen years of mass screening has only kept the problem in abeyance through early identification of afflicted children; it has not wiped out the disease or the causes of the disease. Although pollution due to combustion of leaded gasoline has decreased markedly in the last decade, there are still many dangerous lead sources in children's environments.

Complacency about childhood lead poisoning guarantees a replay of past tragedies when many children were correctly diagnosed only after the onset of convulsions and coma. In 1984, two such cases were brought to my attention. One of the two, a 2-year-old girl underwent brain surgery twice for increased intracranial pressure caused by lead encephalopathy. This is reminiscent of reports in the 1930's of children with lead encephalopathy who were misdiagnosed as suffering from brain tumor.[5]

Today, two decades after childhood lead poisoning first attracted national attention, it is time to re-examine the scope of the problem. This paper will attempt to provide a national perspective on childhood lead poisoning in the United States by reviewing screening and survey data of the last 15 years. It will also examine some common myths and facts about lead poisoning in children; review recent Federal and State activities in preventing this disease under the Maternal and Child Health (MCH) Block Grant; and re-examine the rapidly evolving concept of lead poisoning in children.

Recent Screening and Survey Data

To understand the progress that has been made in the last 15 years, it is important to recall that in the late 1960's and early 1970's, several old cities—Baltimore, Chicago, New York City and Philadelphia—found 25-45 percent of children screened to have blood lead levels of 40 ug/dl or more—the cut-off limit generally used then.[6] Congress, convinced that childhood lead poisoning was a very serious public health problem, passed the 1971 Lead-Based Paint Poisoning Prevention Act. Since mass screening began, despite repeated downward revision of the criteria for a positive case, the percentage of

children with positive findings has declined dramatically as the data below will demonstrate.

CDC Screening Data (FY 72-81)

The 1971 Act authorized Federal financial assistance to help communities develop and carry out detection, treatment and abatement programs for childhood lead poisoning. In FY 72, mass screening and other prevention activities under the Act began. Administered as a categorical grant program by the Centers for Disease Control (CDC), the program expanded from 37 projects in FY 72 to 62 projects in FY 81. In FY 82 the Act, together with several other categorical grant programs, was consolidated into the Maternal and Child Health Block Grant, administered by the Division of Maternal and Child Health (DMCH), Bureau of Health Care Delivery and Assistance (BHCDA), in the Health Resources and Services Administration (HRSA) of the U.S. Public Health Service. From FY 72 to FY 81, projects funded by CDC screened close to 4 million children and uncovered 6 percent to have lead toxicity. The number of children screened by these projects rose from 277,346 in FY 73 to 535,730 in FY 81 while the percentage of children with positive findings reported fell from 11.1 percent to 4.1 percent.[7] (Table I)

TABLE I
Centers for Disease Control Data

FY	No. Screened	% Confirmed Positive
1973	277,346	11.1%
1974	371,955	6.4%
1975	440,904	6.5%
1976	404,818	8.3%
1977	380,496	7.4%
1978	398,701	6.5%
1979	464,751	7.0%
1980	502,925	5.3%
1981	535,730	4.1%

NHANES II Data (1976-1980)

The CDC mass screening data provided useful information only about undue lead exposure in children from high risk neighborhoods because of the targeted nature of the program. The true magnitude of the problem in U.S. children remained unclear until results of the Second National Health and Nutrition Examination Survey (NHANES

II) were released in late 1981. Based on data collected from a probability cluster sample that represented the U.S. civilian non-institutionalized population, the 1976-1980 survey found that 4 percent of children 6 months to 5 years had blood lead levels that exceeded the then accepted limit of 30 ug/dl. It confirmed earlier impressions that the problem of undue lead absorption was nationwide but affected the black, the poor and the central city children disproportionately. Only 2 percent of white but 12.2 percent of black children had elevated blood lead levels. Ten point nine percent of children from families with annual income of $6,000 or less but only 1.2 percent of those from families with income of $15,000 or more had increased blood lead levels. In the low-income population, 18.5 percent of black and 5.9 percent of white children were affected. Of urban children, 7.2 percent compared to 2.1 percent of rural children had a lead problem. In the central cities, 11.6 percent of all children, 18.6 percent of black children, and 4.0 percent of white children were affected.[8] These data, which surprised many, clearly indicated that undue exposure to lead among U.S. children was a leading preventable pediatric public health problem. (Table II)

TABLE II

NHANES II Data (1976-1980)

Percent of Children 6 Months—5 Years with Pb-B Above 30 ug/dl

Demographic Variables	All Races	White	Black
All Children 6 mo.—5 yrs.	4.0	2.0	12.2
Annual Family Income			
under $6,000	10.9	5.9	18.5
$6,000-$14,999	4.2	2.2	12.1
$15,000 or more	1.2	0.7	2.8
Place of Residence			
Urban > 1 million	7.2	4.0	15.2
Central City	11.6	4.5	18.6
Non-Central City	3.7	3.8	3.3
Urban < 1 million	3.5	1.6	10.2
Rural	2.1	1.2	10.3

The NHANES II data became available just as the lead program was consolidated into the MCH Block Grant. Because of the prevalence of the problem, the DMCH in 1982 issued a statement to the regions recommending routine erythrocyte protoporphyrin (EP) screening of all preschool children. This provided a new direction to the States under the MCH Block Grant, i.e. screening should no longer be

limited to high risk groups but should be expanded to include all preschool children. In short, EP screening should be provided as part of routine health care of all preschool children. Since iron deficiency, also common in this preschool population, is detectable by EP before anemia occurs, such routine screening identifies two of the most common preventable health problems in children.

ASTHO Data (FY 82-84)

Beginning in FY 82, with support from the Division of Maternal and Child Health, HRSA, the Association of State and Territorial Health Officials (ASTHO) Foundation began collecting data on childhood lead poisoning prevention activities from State Health Agencies (SHAs). Since submission of information to ASTHO is voluntary, the data are quite incomplete, but there are indications that while many States have expanded their activities under the MCH Block Grant others have yet to initiate any program. In 1982, 26 SHAs reported lead poisoning prevention activities to ASTHO; 552,235 children were screened and 2.0 percent found to have lead toxicity. In FY 83, the reported number of children screened rose to 676,571 and 1.6 percent had lead toxicity. In FY 84, 758,503 children were reported to be screened and 1.1 percent had confirmed lead toxicity (Table III).

TABLE III

ASTHO (PHF) Data on Screening Under MCH Block Grant

FY	No. Screened	% Confirmed Positive
1982	552,235	2.5%
1983	676,571	1.6%
1984	758,503	1.1%

Even though the ASTHO data are incomplete, they give an idea of the current activities and scope of the problem. In FY 84, based on information provided by 10 SHAs, 28.5 percent of children with confirmed lead toxicity underwent chelation therapy. Of children who had environmental investigation, lead hazard was found in 87 percent, and 98.4 percent of the hazardous sources were identified as lead paint. Abatement of lead hazard was reported in 70.4 percent of cases with identified sources[7,9].

Interpretation of Screening Data

The above data provide a broad national perspective on the problem, but the prevalence rate varies widely from State to State and

from area to area in a State. National data often tell little of the situation in a particular area. For example, in FY 83, nationwide the rate of confirmed lead toxicity in the ASTHO data was 1.6 percent but the rate ranged from 0.2 percent in West Virginia to 10.9 percent in Missouri. In the same year, the rate varied from 1 percent in New York City to 9.6 percent in St. Louis; Boston and Chicago both reported a rate of 2.5 percent and Philadelphia had a rate of 3.2 percent (Table IV).

TABLE IV

Screening Data From Selected Cities, 1983

City	No. Screened	% Confirmed Positive
Minneapolis	2,505	0.8%
Baltimore (FY 84)	30,800	0.9%
New York City	120,000	1.0%
Boston	24.402	2.5%
Chicago	34,222	2.5%
Philadelphia	14,400	3.2%
St. Louis	11,736	9.6%

The screening data of the last 15 years suggest a downward trend in the prevalence of undue lead absorption. But in interpreting these data, several facts should be considered. When mass screening began in 1972, blood lead determination was the only screening test used, and a level of 40 ug/dl was used as the cut-off point for a positive case. In 1975, the CDC recommended EP as a simple and cost-effective screening test; an EP level of 60 ug/dl with a blood lead level (PbB) of 30 ug/dl were recommended as criteria for lead toxicity. Most programs quickly began using EP instead of PbB in screening. In 1978, CDC lowered the EP limit to 50 ug/dl without decreasing the PbB level. Because of the change in screening technique and the lowering of cut-off limits for both PbB and EP, data collected during different periods are not comparable. Furthermore, earlier programs concentrated on the children at highest risk. As screening expanded, programs reached out to those at lower risk. Under the MCH Block Grant, routine EP screening of preschool children is recommended. This is likely to increase the number of children screened but further lower the rate of positive findings. In some programs, budgetary constraints have led to a discontinuation or decrease in door-to-door screening, an approach known to produce the highest yield. This also lowers the prevalence rate reported.

In comparing screening under the categorical grant program and the MCH Block Grant Program, it should be noted that some of the screening activities in FY 82 under the MCH Block Grant were supported by funds carried over from FY 81 CDC categorical grants. Moreover, until FY 82, there was no lead screening data collection system other than that of CDC which gathered information only from projects supported by categorical grants, even though some activities existed outside of these projects. The number of children screened between FY 72 and 81 is therefore probably larger than what the CDC data reflect. Likewise, the ASTHO data are very incomplete and should be viewed as minimum figures. For example, a telephone survey in 1984 indicated that lead poisoning prevention activities existed in 40 States, the District of Columbia and Puerto Rico. In FY 84, however, only 26 SHAs submitted data on the number of children screened, 21 SHAs on the number of children with confirmed lead toxicity and 10 SHAs on environmental investigation.[9]

Myths and Facts About Childhood Lead Poisoning

Despite the existence of the above data which clearly documents the magnitude and nature of the problem, childhood lead poisoning remains one of the most poorly understood diseases. Some people erroneously assume that the 1971 Lead-Based Paint Poisoning Prevention Act has legislated the disease out of existence; others mistakenly think that the reduction of lead content of gasoline has completely resolved the environmental lead pollution problem; still others continue to perceive lead poisoning as a disease confined to poor children living in inner city slums. Myths about this disease abound. The following is a summary of the common myths and facts on childhood lead poisoning in the United States.

☆ Myth: Lead paint poisoning is no longer a serious problem today.

Fact: Screening programs have continued to uncover thousands of children with increased blood lead levels each year, and lead paint has remained the most common source of hazard identified. Today, an estimated 40-50 million houses with lead paint remain in use.[10]

☆ Myth: Childhood lead poisoning is caused only by lead paint.

Fact: Lead paint is the major source of poisoning in children. It presents a hazard not only as chips and flakes, but also as housedust and garden soil that are frequently swallowed by young children. There are, however, other sources of lead hazards.

☆ Myth: Lead paint hazard is confined to dilapidated housing.

Fact: Lead paint is also found in expensive housing. Many cases of lead paint poisoning have occurred in well-maintained high-priced housing as a result of exposure

during renovation. These cases are sometimes dubbed as "Yuppie Lead Poisoning" because children from affluent families are the victims.

☆ Myth: Childhood lead poisoning is an urban problem.
Fact: Childhood lead poisoning is more common in urban areas, particularly in old inner cities, but it also occurs in rural areas as the NHANES II data indicated.[8]

☆ Myth: Children must have pica to get lead poisoning.
Fact: Pica contributes to lead poisoning, but normal hand-to-mouth activity common to all young children is also an important factor in lead ingestion. Through such activity, children often swallow toxic amounts of lead dust and soil.

☆ Myth: Lead paint is found only in pre-1940 housing.
Fact: The Consumer Product Safety Commission regulated the lead content of paint for dwellings, toys and furniture only in 1978. A 1975 National Bureau of Standards survey found that 43 percent of housing built between 1940 and 1959 and 13 percent of housing built between 1960 and 1975 had lead paint as defined by an XRF reading of 2 mg/cm^2 or more. Today, the cut-off limit for a positive XRF reading is set at 0.7 mg/cm^2. It is therefore obvious that a sizeable fraction of houses built after 1940 also have lead paint on them.[7]

☆ Myth: Lead paint is no longer manufactured today.
Fact: Lead paint is still manufactured for application other than for dwellings, toys and furniture. In 1986, 14.400 metric tons of lead oxide were used in paint.[1]

☆ Myth: Abatement of lead paint hazard in housing is a reasonably simple procedure, and if done in compliance with local housing codes, assures one of a lead-safe house.
Fact: Many housing codes are quite inadequate and compliance does not necessarily assure one of a lead-safe house. Improper abatement procedures often increase the lead hazard by reducing paint flakes to fine lead particles that are highly absorbable and difficult to remove completely. Following improper abatement procedures the walls may be free of lead paint but the floor, windowsills, carpet and furniture may be full of lead dust.[11]

☆ Myth: Lead in dust and soil comes only from gasoline.
Fact: Lead in gasoline is a major source of environmental pollution, but lead in dust and soil also come from weathering of paint, industrial emissions, incineration and other sources.

☆Myth: Children with lead poisoning will appear sick.

Fact: Most children with lead poisoning or lead toxicity as defined by today's criteria do not have overt clinical symptoms. Because of the difficulty of making a clinical diagnosis of lead poisoning, it is important that children be screened for this disease.

Federal and State Activities Under the Maternal and Child Health Block Grant

In FY 82, with passage of P.L. 97-35, the Maternal and Child Health Services Block Grant Act and the Omnibus Budget Reconciliation Act of 1981, the administrative responsibility for the prevention of childhood lead poisoning under the Lead-Based Paint Poisoning Prevention Act passed from the CDC to the DMCH, BHCDA in the HRSA. The CDC categorical grant program for lead poisoning was terminated. Under the MCH Block Grant, each State sets its own priorities in using the Block Grant funds and determines its budget for lead poisoning prevention. Some States have initiated new statewide or local programs or expanded existing ones; others have continued to view childhood lead poisoning as a health problem of low priority. A 1987 survey by the National Center for Education in Maternal and Child Health indicated that 10 States (Alabama, Alaska, Montana, New Mexico, Nevada, Oregon, South Dakota, Washington, West Virginia and Wyoming) reported no lead poisoning prevention activities.[12]

Since the inception of the MCH Block Grant, the DMCH has supported a number of lead poisoning prevention activities through funds set aside for Special Projects of Regional and National Significance (SPRANS). These include quality assurance programs such as the EP Proficiency Testing Program at the State Lab of Hygiene in Wisconsin, a collaborative study on the standard materials for hematofluorometer, and more recently, after CDC terminated its role, the Blood Lead Proficiency Testing Program; training programs such as the Lead Screening Training Workshop at Louisville, Kentucky and the New England Consortium of Childhood Lead Poisoning Programs; research projects such as the investigation of the loss of essential elements in chelation therapy, the use of X-ray fluorescence (XRF) as a non-invasive tool in measuring bone lead content in children, and the trial of dimercaptosuccinic acid (DMSA) as a new oral chelating agent; data collection on lead poisoning prevention activities from State Health Agencies through ASTHO; information dissemination through the National Center for Education in Maternal and Child Health and the National Maternal and Child Health Clearinghouse; and sponsoring of regional and national conferences on childhood lead poisoning. In addition to supporting these activities, DMCH has also provided consultation and technical assistance to State and local programs,

other federal agencies, and public and private institutions involved in the prevention of lead poisoning.

The Changing Concept of Lead Poisoning

Only twenty years ago, childhood lead poisoning was generally viewed as a clinical disease associated with overt signs and symptoms such as vomiting, irritability, abdominal pain and behavioral changes. Since such manifestations usually become evident at blood lead levels of 60 ug/dl or more, this level became erroneously considered as the upper limit of "normal" blood lead levels.[6] In 1970, the Surgeon General's Statement on the Medical Aspects of Childhood Lead Poisoning defined a blood lead level of over 40 ug/dl as evidence of undue lead absorption which requires investigation into the sources of hazardous exposure.[13] When mass screening of children under the Lead-Based Paint Poisoning Prevention Act began in 1971, 40 ug/dl was used as the cut-off limit for a positive case. In 1972, an article in the New England Journal of Medicine examined in detail the phenomenon of undue lead absorption in children, and raised the question on "whether or not lead causes permanent damage in humans at a low level of absorption and in the absence of clinical symptoms."[6] A number of studies followed which suggested that young children with elevated blood lead levels who had no overt clinical symptoms of lead poisoning did in fact sustain subtle neuropsychological damage.[14,15] In 1975, the Centers for Disease Control lowered the limit of blood lead levels for a case to 30 ug/dl.[16] In the years that followed, many studies questioned the safety of this blood lead level in young children.[17] In 1985, CDC further lowered the definition of elevated blood lead levels to 25 ug/dl but pointed out that such definition should not be interpreted as implying that a safe level of blood lead has been established.[18] In 1986, the World Health Organization recommended a limit of 20 ug/dl while the Clean Air Scientific Advisory Committee of the Environmental Protection Agency suggested a limit of 10-15 ug/dl.[10] The papers presented by Bellinger, Schwartz and Needleman at this conference clearly raise serious questions about the safety of blood lead levels as low as 10-15 ug/dl, particularly for pregnant women and young children.

Today, the concept of lead poisoning extends far beyond the overt clinical manifestations of the disease, to encompass the subtle toxic effects that are demonstratable only by careful psychological evaluation, electro neurophysiological studies, metabolic investigation and statistical analysis of epidemiological data. As more refined research tools have become available in recent years, the toxic effects of this lethal metal have become demonstratable at lower and lower levels. If one acknowledges that lead is toxic at such low levels, then one must also deal with the fact that an enormous fraction of the U.S. population has been affected. In the NHANES II data, 4 percent of preschool

children had blood lead levels that exceeded 30 ug/dl, 9.1 percent had levels that exceeded 25 ug/dl, and 24.5 percent had levels that exceeded 20 ug/dl. Among black children, 52.2 percent had blood lead levels of 20 ug/dl or more.[19] Even though the mean blood lead level of the U.S. population has declined very significantly in the last decade following reduction in the lead content of gasoline,[2] today a sizeable fraction of children still have blood lead levels that exceed 10-15 ug/dl.

TABLE V

Percent of Children 6 Months-5 Years with Blood Lead Above Selected Levels (NHANES II)

	20 ug/dl	25 ug/dl	30 ug/dl
All Races	24.5%	9.1%	4.0%
White	18.1%	5.5%	2.0%
Black	52.2%	24.5%	12.2%

Conclusion

The extent of damage lead has caused in our children is beyond estimation. Currently, the pressing issue in childhood lead poisoning is not defining a toxic level, since there may be no threshold for the toxic effects of lead in the young. Attention must be focused on the prevention of further environmental pollution by this lethal element that is already ubiquitous in our modern world, application of proper abatement techniques in dealing with existing hazards, and minimizing exposure among young children and pregnant women. The prevention of childhood lead poisoning should no longer by aimed only at the prevention of mental retardation and learning disabilities but at the prevention of any damage, no matter how subtle, that may compromise the optimum mental and physical growth, development, and function of the human being.

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LEAD POISONING, HOUSING, AND JOBS

Herbert L. Needleman, M.D.

Childhood lead poisoning is a man-made disease. Lead's sources, the pathways through which it enters children's bodies, and its effects on their brains are no longer a mystery. Why then has our society been so sluggish in eliminating this problem? Two myths surround the disease; both are partly responsible. The first is that lead afflicts only poor American children, and that in some way, inferior parental care is at the root. The second myth is, paradoxically, that the disease has already been conquered, that recent regulations governing the amount of lead in household paint and gasoline have virtually eliminated the sources for human exposure. Both beliefs have been severely damaged by new scientific data.

Most data on lead toxicity has come from studies of American children. This September in New Orleans, at an international meeting of trace metal and health scientists, an impressive body of new information on these questions was presented. Studies from Denmark, Greece and Scotland showed clear evidence that children's IQ scores were reduced by very small elevations in tooth or blood lead. The children in these studies were not selected from the ranks of the poor; this finding effectively destroyed the first myth, and settled to the satisfaction of all but the lead industry's representatives, the contentious question of low dose lead effects on children's IQ scores. IQ deficits were observed at blood levels of 15 ug/dl (parts per hundred million), well below the current U.S. Centers for Disease Control (CDC) standard of 25 ug/dl.

Lead's mischief is not limited to IQ scores. Hearing is impaired at blood lead levels as low as 15 in two separate investigations. Lead exposure slows physical growth in infants in the first year of life, and through the primary grades. Lead crosses the placenta, and has been measured in infants' umbilical cord blood at birth. Boston newborns demonstrated lead-related increases in the risk for minor congenital anomalies. Two years later, these infants showed a strong relationship between prenatal lead level and intelligence test scores.

These investigations draw a convincing picture of lead's broad devastations on children's intelligence, growth, and language perception. These effects begin at levels well below 25 ug/dl, the current Centers for Disease Control defined toxic threshold. The CDC has regularly revised this standard in response to the latest data. It is clearly time for another look at the current value.

With regard to the second myth, lead levels in children and adults have in fact declined in recent years. Lead screening programs, infant

nutritional supplements and the removal of lead in gasoline have acted in concert to achieve this result. But it is clear that truly frightening numbers of children are in the range that now must be defined as hazardous. Approximately 2.5 million American children are at risk.

The three most important sources of lead for children are air, water, and old paint. Hard-won EPA regulations have reduced lead in gasoline in stepwise fashion. Blood lead levels in adults, children and newborns have dropped in close correlation. A recent EPA document estimates that 16% of drinking water supplies have levels of lead above standard. Identification of the source of the hazard, and appropriate steps to reduce lead in drinking water have been called for, and require urgent address.

Although lead in household paint was banned by statute in 1972, as many as 24 million homes still have surfaces painted well before then. There are approximately 2 million homes in the U.S. with deteriorated lead painted interiors in which young children live. This paint, containing as much as 50% lead by weight, blisters and flakes, or simply powders and becomes part of the household dust. There the dangerous residue awaits the daily explorations of the curious child. While lead exposure is not limited to Americans or the poor, the poor every day come into contact with more lead. This excess of lead in paint is tightly bound with two serious shortages: affordable housing and decent jobs. To confirm this, one has just to drive through the ghettoes of North Philadelphia, Chicago or Washington and count the decayed houses, the legions of job hungry men hanging around, shooting baskets, killing time.

Deleading and repainting an average sized home costs $5000. The costs of deleading 2 million homes would be 10 billion dollars. We need plans to train employable persons in deleading, painting, and housing rehabilitation in their own neighborhoods. Thousands of real jobs, at decent pay levels can be created, and houses made safe and livable. The money earned by these men would be spent in the neighborhood shops where they live, and the impact multiplied as the money circulated.

Smallpox was once considered a biological given. It is gone. Like smallpox, lead paint poisoning can be—not simply reduced or controlled—it can be wiped out forever. A serious attack on lead is simultaneously an attack on three, not one, man-made and therefore man-curable diseases.

RECENT STUDIES OF THE DEVELOPMENTAL AND NEUROPSYCHOLOGIC EFFECTS OF LOW-LEVEL LEAD EXPOSURE

David Bellinger, Ph.D.

Introduction

In the late 1970's, the landmark study by Needleman, Gunnoe, Leviton et al. (1979) provoked widespread debate on whether so-called "asymptomatic" or "subclinical" lead levels impair children's cognition and thus limit their academic achievements. In that study, less optimal neuropsychologic function and classroom behavior were noted among children with levels of lead in their shed deciduous teeth that were high with respect to average exposure, but not sufficient to elicit classical signs or symptoms of lead poisoning. These findings raised the spectre of a "silent epidemic" of lead-associated learning disability among urban children and were to a large extent responsible for the impressive volume of research conducted in the ensuing decade on the reproductive, developmental, and cognitive toxicity of lead at doses within what is considered to be the "normal" range (i.e., "low-level" lead exposure). Some studies focused on preschool or school-age children using a retrospective or cross-sectional design similar to the one used by Needleman et al. (e.g., Yule et al., 1981; Ernhart et al., 1981; Smith et al., 1983; Winneke et al., 1983, 1985, 1987; Hatzakis et al., 1985, 1987; Fulton et al., 1987; Raab et al., 1987; Hansen et al., 1987). Others are prospective, involving frequent assessment of the lead exposure and development of cohorts assembled at or even prior to birth. This paper will focus on these prospective studies, in particular describing how use of this design has permitted the investigation of certain issues difficult to address by retrospective or cross-sectional methods. Despite certain inherent limitations, however, retrospective and cross-sectional studies continue to provide valuable insights on lead toxicity. The scrutiny given to each new study has motivated substantial increases in the rigor with which investigators approach epidemiologic issues such as selection biases, and biostatistical issues such as confounding.

Advantages of a Prospective Study which Begins at Birth

The delineation of critical periods of exposure, i.e., variations in vulnerability within childhood, is requisite for the formulation of policy that adequately protects the most vulnerable subgroups of the population. Serial measurements of blood lead level provide the detailed histories of children's lead exposure needed to identify such

periods. In particular, prospective studies permit investigation of lead's behavioral teratogenicity, i.e., the hazards of *in utero* exposure. Studies of occupationally-exposed women and men and case studies of maternal intoxication during pregnancy have shown that high-dose prenatal exposure is associated with very poor fetal outcomes (Bellinger & Needleman, 1985). However, the prospective studies represent the first population-based assessments of the sequelae of relatively low-level prenatal exposure.

With the availability of detailed exposure histories, inferences about the threshold of effect can also be made with greater certainty. Because of the limited historical information conveyed by a recent blood lead level or even an index of cumulative exposure such as tooth lead level, the level at the time a cognitive deficit is measured may not reflect the level that was responsible for the CNS damage that underlies the deficit. It is this prior level that should serve as the exposure limit, not the current level, which may be either an underestimate or an overestimate.

Finally, because surveillance of the children typically begins at or prior to birth, the prospective studies provide the opportunity to evaluate "reverse causation," a persistent problem in interpreting the results of cross-sectional and retrospective studies. Due to the difficulty of characterizing children's past lead exposure, the early course of their development and, by extension, the temporal relationships between them, investigators have been unable to rule out completely the possibility that children with pre-existing impairment engage in behaviors that cause them to suffer greater lead exposure. In this scenario, increased exposure and neuropsychologic deficit are associated, but not because lead produced the deficit. In principle, whether elevated exposure precedes or follows impaired function can be established with greater certainty in prospective studies.

The purpose of this paper is to summarize the major conclusions our group has drawn from a prospective study we began in 1979. Reference will be made to other prospective studies to illustrate the coherence of the results reported to date, despite marked difference among studies in the populations sampled and patterns of lead exposure. Starr (1985) compares the methodologic features of these studies, while Davis and Svensgaard (1987) integrate, critique and interpret the findings. Finally, general comments will be made about the reversibility of lead-associated deficits, age-related changes in vulnerability, effect modifiers, the distribution of susceptibility in the population, and the public health implications of lead's apparent effects on children's cognition.

Recent Prospective Studies

The Boston Study

We recruited our sample from a group of approximately 12,000 babies born over a two year period. Eligibility was based on the con-

centration of lead in umbilical cord blood. In assembling the cohort, we over-sampled infants with cord blood lead levels below the 10th percentile for this population ("low," <3 ug/dl) as well as those with levels above the 90th percentile ("high," > 10 ug/dl). To provide the opportunity to characterize more precisely the nature of any dose-effect relationship between infants' lead levels and their subsequent development, we also recruited a group of infants with levels close to the mean for this population ("medium," 6 to 7 ug/dl). In total, 249 infants were

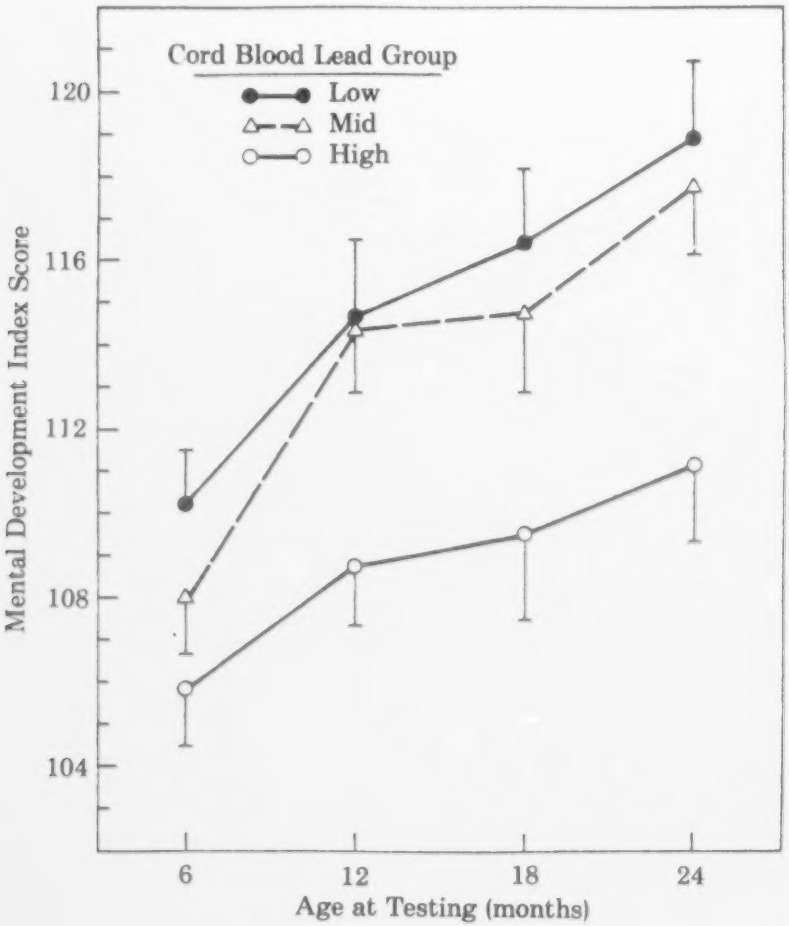

FIGURE 1. Least-squares mean MDI scores through age 24 months for infants classified by umbilical cord blood lead group. Scores are adjusted for 12 potential confounders.

enrolled (85 with low, 88 with medium, 76 with high levels). The highest cord blood lead level of any infant in the sample was 24.9 ug/dl, just below the level currently used by the U.S. Centers for Disease Control (1985) and the American Academy of Pediatrics (1987) to identify children with lead toxicity. Two-thirds of the infants in the high lead group had levels of 10 to 15 ug/dl.

To date, we have assessed the children's postnatal lead exposure and their development at 6, 12, 18, 24, and 57 months of age. At the 4 younger ages, the Bayley Scales of Infant Development (Bayley, 1969) were administered and capillary blood samples collected. At age 57 months, the McCarthy Scales of Children's Abilities were administered and venous blood samples collected. Briefly stated, infants' performance on the Bayley Scales, specifically the Mental Development Index (MDI), between 6 and 24 months of age was strongly related to their cord blood lead levels (Bellinger et al., 1987a). On all occasions, infants with high cord blood lead levels (i.e., 10 to 24.9 ug/dl) scored 4 to 8 points lower than infants with cord blood lead levels in the low or medium ranges (Figure 1). The performance of the infants in these latter ranges was comparable. Because differences among exposure groups in terms of factors other than lead could be responsible for these performance differences, we took account of the following variables when evaluating the contribution of cord blood lead level to children's MDI scores: maternal age, IQ and education, family social class, race, alcohol consumption during pregnancy, smoking history, quality of the rearing environment provided for the child, infant sex, birthweight, gestational age, birthorder. Because of the unusual associations between lead and these potential confounders in our predominantly middle and upper-middle class sample (Bellinger, Leviton, Waternaux, and Allred, 1985), adjustment for these factors tended to increase rather than reduce the strength of the association between lead and MDI scores (Bellinger, Leviton, Waternaux, Needleman, and Rabinowitz, 1986b).

At most ages, the mean postnatal levels of the infants in the three prenatal exposure groups did not differ significantly. In other words, the infants with high prenatal exposure displayed performance deficits through two years of age even though, by age 6 months, their blood lead levels had fallen to what might be considered "control" levels (i.e., <10 ug/dl). Postnatal blood lead levels were not associated with infants' MDI scores, whether levels at the various ages were considered separately, combined into an index of cumulative postnatal exposure, or combined with cord blood lead level to test for a synergistic effect of prenatal and postnatal exposure on early development.

Other Prospective Studies

Similar associations have been noted in a sample that differs from ours sociodemographically and in the magnitude of postnatal lead exposure. Several hundred lower-class, mostly minority women were

recruited from areas of Cincinnati in which pediatric lead poisoning is prevalent. Blood samples were obtained from women at the time of the first prenatal visit (6 to 28th week of pregnancy). The prenatal blood lead levels ($\overline{X}$=8.0, range: 1 to 27) were comparable to the umbilical cord blood lead levels of the infants in our sample. Mean blood lead levels of the children remained below 7 ug/dl through the newborn period (measured at 10 days and at 3 months), although they increased two-fold, to 14 ug/dl, by the end of the first year (Bornschein et al., 1985). Both prenatal and umbilical cord blood lead levels were negatively associated with infants' performance on the Bayley Scales (MDI) at 3 months of age (Dietrich et al., 1987a, 1987b). The reduction in MDI scores, approximately 6 points for each 10 ug/dl rise in cord blood lead level, is comparable to the reduction we observed in our more socioeconomically-advantaged sample. Prenatal, newborn (10 day), and to a lesser extent umbilical cord blood lead levels were significantly associated with MDI scores at 6 months of age. Preliminary analyses (Dietrich et al., 1986) reveal that MDI scores at 12 months are related to prenatal lead exposure, but "indirectly" via a lead-associated reduction in birthweight (Bornschein et al., 1987). As in our study, no associations have been observed between infants' early postnatal blood lead levels and their scores on the Bayley Scales.

Winneke et al. (1985) reported that indices of perinatal exposure explained nearly as much variance in the reaction time performance of school-aged German children as did current blood lead level.

Not all the prospective studies have observed this association between low-level prenatal lead exposure and early development. McMichael et al. (1986) reported a mean blood lead level of approximately 10 ug/dl in a cohort of pregnant women living in proximity to a large lead smelter in Port Pirie, South Australia. Prenatal and umbilical cord blood lead levels bore no relationship to infants' MDI scores on the Bayley Scales at 2 years of age (Baghurst et al., 1987). As Davis and Svensgaard (1987) note, however, the lead levels of the children in this heavily contaminated area increased substantially after birth. The mean level remained above 20 ug/dl between the ages of 15 and 36 months, and above 15 ug/dl through 5 years of age. This may have obscured an association between prenatal lead levels and early development, especially in view of the fact that the Bayley Scales were not administered until the children were 2 years of age.

The absence of a consistent pattern of associations between prenatal lead exposure and development has also been reported in a series of studies by Ernhart and colleagues. Umbilical cord blood lead levels, which averaged 5.8 ug/dl and ranged from 2.6 to 14.7, were significantly associated with scores on the abnormal reflexes scale of the Neonatal Behavioral Assessment Scale (NBAS) and with scores on the neurological soft signs scale of the Graham/Rosenblith Behavioral Examination of the Neonate (G/R) (Ernhart et al., 1986). Moreover, soft signs score significantly predicted MDI scores at one year of age (Wolf, Ernhart, & White, 1985). However, maternal blood lead levels at

delivery and infant cord blood lead levels were not significantly associated with several other scales of the NBAS or the G/R, MDI scores at 6 months, 1 year, or 2 years, Stanford-Binet scores at 3 years (Ernhart, Morrow-Tlucak, Marler & Wolf, 1987), language development at age 3 years (Morrow-Tlucak & Ernhart, 1987), or WPPSI scores at age 4 years 10 months (Ernhart & Morrow-Tlucak, 1987). Half the mothers recruited into this sample had a history of alcohol abuse. In view of the evidence that alcohol is a behavioral teratogen (Streissguth & LaDue, 1987), this competing exposure may have impeded identification of any variance in infants' performance that is attributable to lead exposure.

Issues in Characterizing Lead's Neuropsychologic Toxicity

Critical Periods of Exposure

The early findings of the Boston and Cincinnati prospective studies are strikingly consistent in suggesting that the fetus may be more sensitive than is the young child to low-level lead toxicity. At present, however, the long-term significance of the early developmental deficits noted in the children with "high" prenatal exposures is far from certain. Preliminary analyses of the scores achieved by the children in our sample at age 5 years on the McCarthy Scales of Children's Abilities suggest that these effects of prenatal exposure are reversible (Bellinger et al., 1987b). The General Cognitive Index (GCI) scores of children in the three prenatal exposure groups did not differ significantly from one another. If postnatal exposure is low, adverse cognitive effects of prenatal exposure may attenuate over a period of years (in this case, within the interval 2 to 5 years after the "high" exposure).

Recent results from several studies of school-aged children and from preschool evaluations of the prospective cohorts suggest that certain aspects of children's behavior and cognition may, indeed, be impaired by postnatal exposures corresponding to blood lead levels in the upper reaches of the 0 to 25 ug/dl range. Although the General Cognitive Index scores achieved at age 5 by the children in our study were not significantly associated with prenatal exposure, they were associated with postnatal blood lead levels, especially those measured at 2 years of age (Bellinger et al., 1987b). The mean postnatal levels in this sample never exceeded 8 ug/dl and in only a few cases did individual values exceed 20 ug/dl.

In the cohort of Port Pirie children, the strongest associations found to date are between postnatal lead levels and MDI scores. Preliminary analyses suggest that postnatal lead levels are significant predictors of children's scores on the McCarthy Scales at age 4 (Baghurst et al., 1987). In a large-scale study of 6 to 9 year old Scottish children with a mean blood lead level of 11.5 ug/dl (range 3.3 to 34.0), Fulton et al. (1987) observed a dose-dependent association between lead level and performance on the British Ability Scales. In another

UK study, Yule et al. (1981) noted that children with blood lead levels between 13 and 32 ug/dl achieved significantly lower scores on the WISC-R than did children with lead levels below 13 ug/dl. Similarly, Winneke et al. (1987) observed significant associations in a sample of German school children between blood lead level ($\bar{X}$=8.4, range 4.4 to 22.8 ug/dl) and both visual-motor integration, and reaction time. Hatzakis et al. (1987) noted increased reaction time latencies and error scores among children with blood lead levels of 15 to 24.9 ug/dl, relative to children with levels less than 15 ug/dl. These data suggest that age-related vulnerability to lead toxicity may not be as striking as the early results of the prospective studies initially suggested.

Relative Vulnerability of Cognitive Functions

In all likelihood, a comparison of exposure groups in terms of global indices such as full-scale IQ is not the most sensitive test of the association between lead and children's neuropsychologic function. Deficits in attentional performance, measured by teacher ratings and by laboratory tasks such as reaction time, have consistently been associated with higher exposure (Needleman et al., 1979; Hunter et al. 1985; Winneke et al., 1983; Hatzakis et al., 1985, 1987; Silva et al., 1988). In many studies, the dose-response relationship extended to lower levels of exposure for these outcomes than for more traditional measures of intelligence.

Earlier studies tended to identify verbal skills as the most sensitive outcomes (Mayfield, 1983). Some recent studies also report lead to be associated with linguistic outcomes ranging from WISC-R verbal IQ score (Hatzakis et al., 1987; Hansen et al., 1987) to reading achievement (Fulton et al., 1987). In other recent studies, however, the domains in which the most highly exposed children have greatest difficulty are visual-motor integration and visuo-spatial skills (e.g., figure reproduction tasks) (Hansen et al., 1987; Winneke et al., 1987). In our prospective study, the infants with higher prenatal exposure had greater difficulty in their early months with the development of visually-directed reaching and prehension skills (Bellinger et al., 1984; 1986). In later infancy, this was expressed as difficulties in block building, completing simple formboards, and in perceiving part-whole relationships. At age 5, the children with higher postnatal exposures at age 2 performed less well on figure reproduction tasks and jigsaw puzzle completion (Bellinger et al., 1987b).

One explanation for the inconsistency in the associations between lead exposure and function in different domains is that the specific effects of lead may differ depending on the timing of exposure (Shaheen, 1984). Winneke et al (1985b) noted that attentional performance was most strongly associated with level of current exposure, while visual-motor integration was most strongly associated with cumulative exposure. The effects may also depend on the types and amounts of cognitive and psychosocial supports available to a child. For example,

verbal skills may be less affected in children of academically-oriented parents who encourage the development of such skills. As increasingly sophisticated neuropsychologic assessments are applied to samples for whom detailed exposure histories are available, more definitive conclusions can be drawn about the relative vulnerability of different aspects of cognition to lead exposure at different ages in children reared in environments that vary in amounts and types of developmental supports.

Effect Modification

As we collect additional data on our sample, we appreciate more and more the complexity of the interplay between exposure parameters and the context within which exposure occurs. For instance, some patterns we've noted correspond conceptually to 4-way interactions, involving age at lead exposure, dose, age at outcome assessment, and social class (Bellinger et al., 1987c). Among lower-class infants, Mental Development Index scores in the second year of life tended to be lower than expected when prenatal exposure was either "medium" (i.e., 6 to 7 ug/dl) or "high" (i.e., 10 to 25 ug/dl). In contrast, among upper-class infants, scores tended to be lower only when prenatal exposure was high. The pattern differed in two respects when blood lead level at 6 months of age was substituted for cord blood lead level as the basis of exposure classification. During the second year of life, the MDI scores of lower-class infants appeared to be lower only when blood lead level at 6 months of age was high, while the scores of upper-class infants were not associated with the blood lead level measured at 6 months of age. These findings suggest that prenatal exposures are more harmful than are the same levels of exposure suffered in the early postnatal period, at least with respect to MDI scores achieved in the first two years. In addition, lower-class infants may be more vulnerable to low-level lead exposure than are upper-class infants. This observation is consistent with those made by others (Winneke & Kraemer, 1984; Harvey et al., 1984; Dietrich et al., 1987b). In the Cincinnati prospective study, the MDI scores achieved by lower-class infants at 6 months of age declined 16 points across the range of newborn lead levels represented in the sample (1 to 22 ug/dl).

Another factor associated with the likelihood that a child will manifest lead-associated deficit is gender, with males at greater risk than females (Pocock, Ashby, & Smith, 1987; Dietrich et al., 1987b). The MDI scores of male infants in the Cincinnati cohort declined more than 22 points over the 26 ug/dl range (1 to 27) of prenatal blood lead levels.

Distribution of Suspectibility in the Population

The population impact of lead's cognitive toxicity at low doses will depend, in part, on the prevalence of "responders" i.e., the fraction of children whose performance is affected at a given level of exposure.

The clinical picture associated with a given blood lead level varies greatly among children. Individual differences in the sensitivity of children's cognitive function to low dose exposure may be obscured when the statistical methods used are based on comparison of group means. A 4 to 8 point deficit in the scores of children with higher lead levels may be due to a small but consistent deficit in the performance of a large majority of these children. Alternatively a small subset of children (the "responders") may achieve extremely poor scores and exert disproportionate influence on the group mean, while the rest achieve scores comparable to those of children with low exposures. Although the distribution of scores in the high lead group would be bimodal in the first scenario and unimodal in the second, the standard deviations of the two distributions would not necessarily differ. Clearly, however, the public health import would. If most children are "responders," preventive efforts might best be focused on source abatement. If a small percentage are "strong responders" but most are "nonresponders," limiting the exposure of the "responders" may represent the most effective approach to prevention.

Although individual variability in response is often discussed in the context of animal studies, it generally has not entered into the interpretation of human studies (Weiss, 1980). We took a first step, examining the relationships among level of MDI scores, individual variability in scores over time, and level of prenatal lead exposure. An infant's values on the 12 variables considered to be potential confounders were used to calculate the "expected" MDI scores at each of the 4 ages at which the Bayley Scales were administered. In other words, these are the scores we predicted that a child would achieve

TABLE I

*Classification of Infants According to Cord Blood Lead Group and Number of Ages at Which Observed Mental Development Index Score Exceeded Expected Score**

Cord Blood Lead Group	Number of Ages Observed MDI Exceeded Expected MDI					
	0	1	2	3	4	Total
low	5 (8.5)**	9 (15.3)	18 (30.5)	20 (33.9)	7 (11.9)	59
mid	4 (6.5)	12 (19.4)	22 (35.5)	17 (27.4)	7 (11.3)	62
high	13 (23.6)	16 (29.1)	13 (23.6)	8 (14.5)	5 (9.1)	55
total	22	37	53	45	19	176

*expected MDI score at each age was calculated using a regression equation consisting of the 12 variables considered to be potential confounders.

**row percentages

given his birthorder, race, social class, maternal IQ, sex, etc. A summary score, ranging from 0 to 4, was assigned to an infant based on the number of ages at which his or her observed MDI score exceeded the expected score. For instance, a summary score of 4 was assigned to the child who achieved an MDI score higher than the one expected on all four occasions.

Nearly one-fourth (23.6%) of infants with cord blood lead levels of 10 to 25 ug/dl achieved a summary score of 0, indicating that on no occasion did they score higher than expected (Table 1). The corresponding percentages for infants with low and medium prenatal exposures were 8.5 and 6.5, respectively. Among infants with high exposures, the modal summary score was 1 (29.1%); among infants with medium exposures, it was 2 (35.5%); among infants with low exposures, it was 3 (33.9%). Our finding that 53% of those with high exposures scored better than expected on, at most, only one occasion suggests that the lower mean MDI scores of this group are not largely due to the poor performance of a small subset of infants extremely vulnerable to lead. Rather, over the first two years of life, the performance of most of the infants in this group was consistently below the level expected.

This analysis is not directly affected by the absolute level of an infant's performance. In a sense, an infant serves as his or her own control. An infant who consistently achieved scores of 125 might still receive a summary score of 0 or 1 if the circumstances of that infant's life are so favorable that he or she would be expected to score higher. Thus the fact the infants in high exposure group achieved scores well above the standardization sample of the Bayley Scales should not be taken as an indication that lead is without effect at the doses studied.

The "Practical Significance" of the Association

In the analyses summarized above, the performance of an infant is characterized simply as above or below expected. The absolute deviation from the expected score is not taken into account. Such information would bear on other issues besides individual variability in toxic response. Commentators on the literature relating lead exposure and neuropsychologic outcome often note that the 4 to 8 point deficit in the mean IQ scores of the more highly exposed children is equivalent to the standard error of measurement of most IQ tests and that, in any case, a deficit of this magnitude is unlikely to affect how a child functions in a natural setting. Rutter (1980) has pointed out the error in statistical reasoning that underlies the interpretation of group differences in terms of individual performance, while Needleman, Leviton, and Bellinger (1982) illustrate the implications of making this error using the data of Needleman et al., (1979). While the children with high dentine lead levels achieved verbal IQ scores that averaged only four points lower than the scores of children with low dentine lead levels, the frequency of extremely low scores (<80) was 3-fold higher

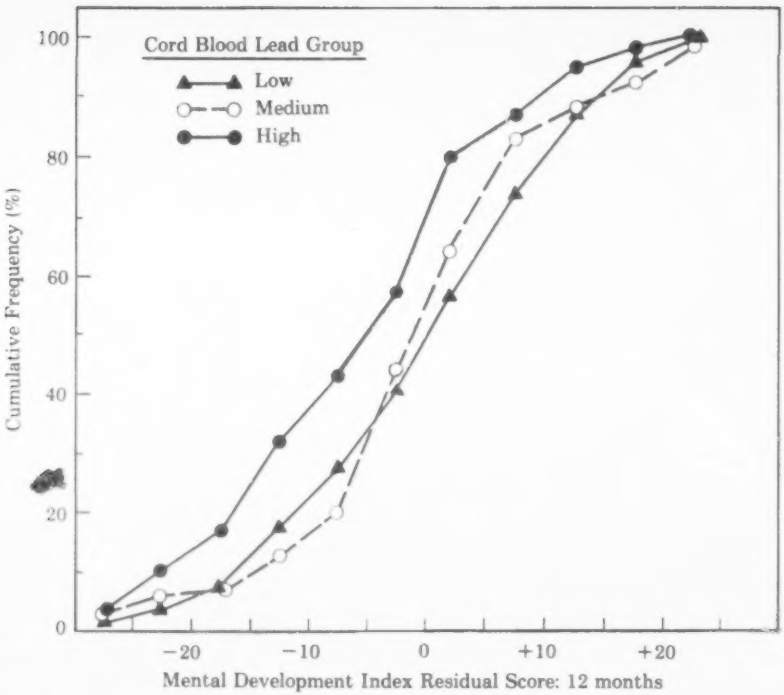

FIGURE 2. Cumulative frequency distributions of residualized 12 month MDI scores for infants classified by umbilical cord blood lead group. Residuals were obtained by regressing MDI scores on 12 potential confounders.

among these children. Conversely, the frequency of extremely high scores (> 125) was much lower in this group.

Using the data from our prospective study, we explored this issue, determining for each prenatal exposure group the cumulative frequency distribution of the amount by which the MDI scores the infants achieved deviated from expectation. In statistical terms, these deviations are the residuals of the regression of MDI scores on the 12 potential confounders. We present these data for MDI scores at 12 and 24 months (Figures 2 and 3). At both ages, the distribution of deviation scores for the high lead group is less sigmoid than are the distributions of the other two groups. The more rapid rise in cumulative frequency indicates that a higher percentage of the infants in this group achieved MDI scores considerably below expectation. Whatever criterion is chosen to identify significant deficits in performance (e.g., more than 10 points below expected, more than 20 points), the prevalence is at least 2-fold higher among the high lead children at both ages. In contrast, the differences between the median residual scores of the three

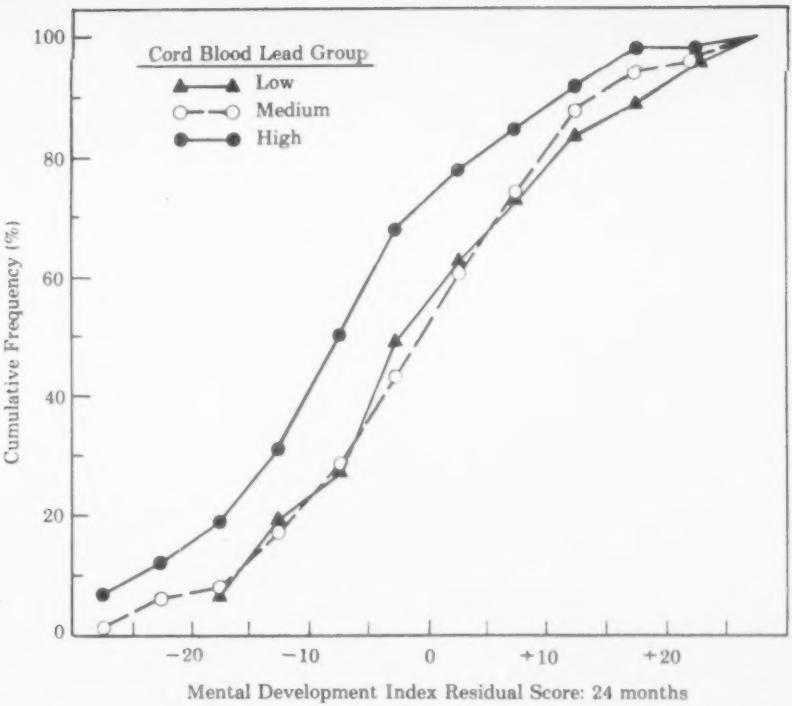

FIGURE 3. Cumulative frequency distributions of residualized 24 month MDI scores for infants classified by umbilical cord blood lead group. Residuals were obtained by regressing MDI scores on 12 potential confounders.

groups (read off by extending a horizontal line from the 50% mark on the ordinate), are modest: 1.4, 0.6, and −1.5 for MDI at 12 months for the low, medium, and high exposure groups, respectively; and 0.0, 2.4, and −5.4 for MDI at 24 months for the low, medium, and high exposure groups, respectively.

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LOW LEVEL HEALTH EFFECTS OF LEAD: GROWTH, DEVELOPMENTAL, AND NEUROLOGICAL DISTURBANCES

Joel Schwartz, Ph.D.

I. EFFECTS OF LEAD ON THE GROWTH AND DEVELOPMENT OF CHILDREN

While cognitive effects have long been central to the field of lead toxicity, several recent studies have examined the effects of low level lead exposure on the growth and development of children. Simultaneously, increased attention has been given to the fetus as a target of low level lead exposure. The effects studied include stature, growth rates, hormonal metabolism, and heme synthesis in children, and birthweight, gestational age, and congenital anomalies in the fetus. The overall pattern of these studies suggests that any threshold for the effects of lead on the fetus or young children is so low as to be inconsequential. While these effects are far less severe than the encephalopathy present in acute lead poisoning, the blood lead levels at which they occur indicate that large segments of the population are effected. Programs aimed solely at preventing high level exposure run the risk of allowing important, although more moderate, health impairment to occur in orders of magnitude more children than those suffering the profound disturbances of high level lead exposure. These new findings are discussed in more detail below.

Epidemiological Studies

Correlation Between Lead and Stature

Short stature has been associated with lead poisoning since the 1920's in Australia,[1] and more recently in asymptomatic cases in the United States.[2,3] However, these studies focussed primarily upon relatively high levels of lead exposure.

In early 1986, Schwartz et al.[4] published cross-sectional analyses of data from the second National Health and Nutrition Examination Survey (NHANES II), showing a relationship between children's blood lead levels and their stature. This survey was a representative sample of the U.S. population, and the study covered 2,695 children aged 6 months to 7 years. Nutritional intake was obtained from a diet recall and a nutritional data bank. This allowed control for 15 nutritional factors, as well as hematocrit, transferrin saturation, and socioeconomic factors. This relationship is illustrated in Figures 1 and 2, after controlling for all the other significant variables.

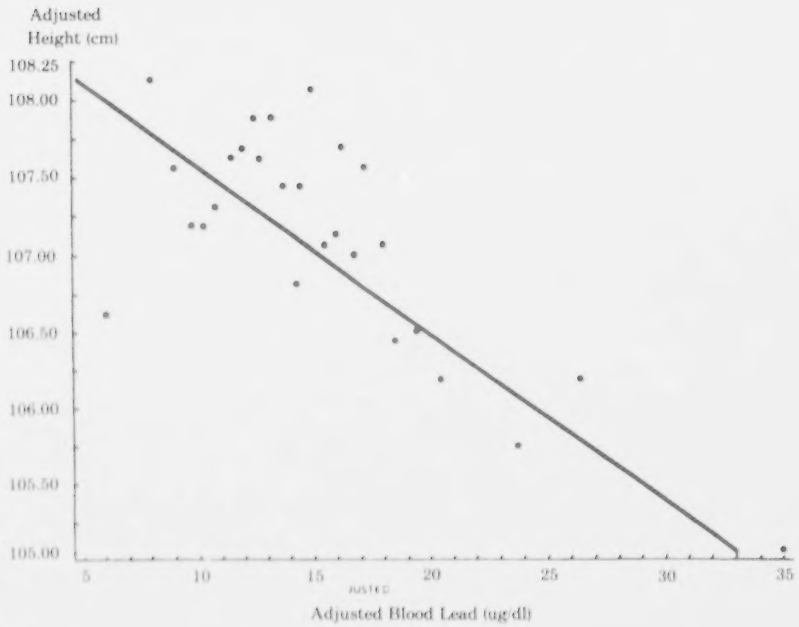

FIGURE 1. Adjusted height and adjusted blood lead levels for children aged 7 years and younger in Second National Health and Nutrition Examination Survey. Both height and blood lead level have been adjusted by regression for effects of age, race, sex, and all other variables at .05 level. Each point is mean height and mean blood lead level of approximately 100 consecutive observations, ordered by blood lead levels. Regression line reflects slope of coefficient obtained from multiple regression analysis of all 2,695 observations.

No threshold for the relationship was found down to the lowest observed levels of blood lead (4 ug/dl). At the mean age of the children studied (59 months), the mean blood lead level of the children was associated with a reduction of about 1.5% below the height that would be expected if their blood leads had been zero. At 25 ug/dl, a reduction in height of about 3% appeared to have occurred. By itself, a cross-sectional epidemiological study cannot definitely establish a causal link. However, these findings have stimulated longitudinal studies as well as animal experiments, that are discussed below, that together form a relatively consistent picture.

Correlation Between Lead and Birth Weight

The association between lead and stature may well begin with fetal exposure. A number of recent studies have addressed this issue. Dietrich et al.,[5] reporting on the Cincinnati prospective study of lead

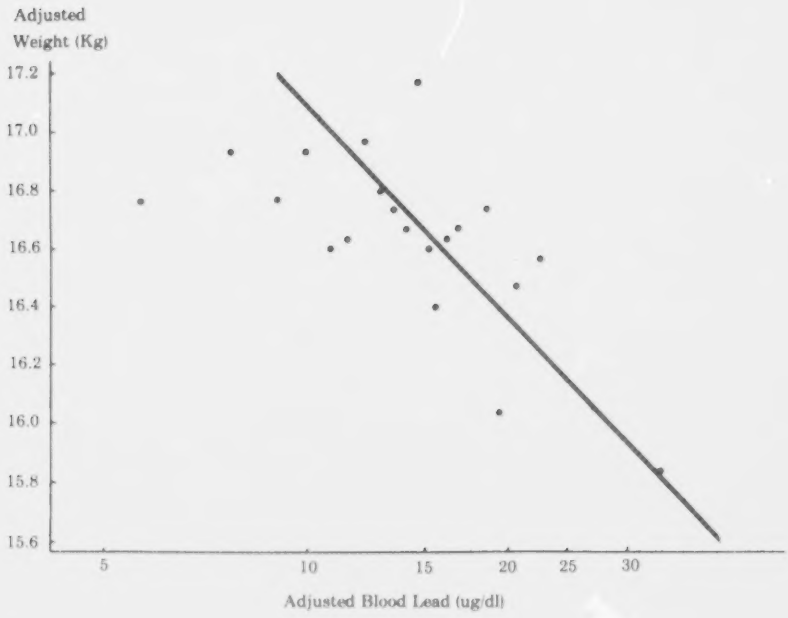

FIGURE 2. Adjusted weight and adjusted blood lead levels for children aged 7 years and younger in Second National Health and Nutrition Examination Survey. Both weight and blood lead level have been adjusted by regression for effects of age, race, sex, and all other variables significant at .05 level. Each point is mean weight and mean blood lead level of approximately 70 consecutive observations, ordered by blood lead levels. Regression line reflects slope of coefficient obtained from multiple regression analysis of all 1,967 observations with no missing data.

exposure, found an association between maternal blood lead levels during pregnancy and reduced birth weight. They also found an association between maternal blood lead levels and reduced gestational age. By the use of structural equation modeling, they were able to demonstrate that the effect of lead on birth weight was both indirectly, through the reduction in gestational age, and also had a direct component, after controlling for gestational age. Recently, Bornschein et al.[6] have reported further analysis of this data. The new results confirm the earlier findings, and add the interesting additional observation that the negative effect of maternal alcohol use on birth weight is not additive with lead. The total effect exceeds the effect of either exposure but by less than an additive amount. They have hypothesized that in nutritionally sufficient pregnancies, there is a natural limit to the negative impact of environmental factors, producing the nonlinearity.

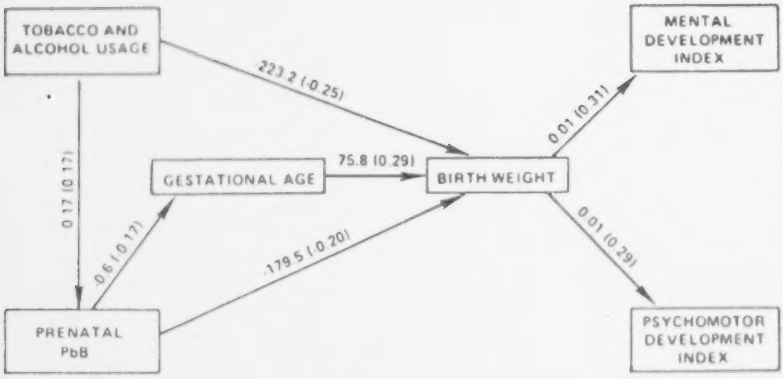

FIGURE 3. Relationships among variables affecting 6-month MDI and PDI scores, as revealed through structural equation analyses. Arrows represent hypothesized relational pathways, with covariate-a usted parameter estimates (and standardized regression coefficients) indicated for each. All relationships are significant at $p \times 0.05$ (one-tail test). Source: Dietrich et al. (1986).

Figure 3, taken from Dietrich et al., shows the relationships they found. The numbers shown for each pathway are the regression coefficients, with their standard errors in parentheses. Thus, a change of 1 in blood lead on the log scale (e.g., from 4 to 8 ug/dl) was associated with a 0.6-week decrease in gestational age. That decrease produced an estimated 46 gm decrease in birth weight, and in addition, a 180 gm reduction in birth weight was associated with that lead exposure through the direct route. The mean maternal blood lead level in this cohort was 8.3 ug/dl, with a standard deviation of 3.8 ug/dl, so these effects are occurring at extremely low blood lead levels.

Bryce-Smith[7] has recently reported an association between both birth weight and head circumference and placental lead levels in a cohort of 100 normal infants. Ward et al.[8] have recently reported more detail from this study. Placental lead levels in children with birth weights under 2500 gm were almost triple those in children above 4000 gm. Placental lead correlated better than maternal blood lead levels with these outcome.

McMichael et al.[9] have reported the results of a prospective study in Australia, in which they found a higher incidence of birth weights under 2500 gm in their high lead area than in their low lead area. However, blood lead levels at delivery were actually lower, although insignificantly so, in the mothers of the low birth weight children. One possible explanation of this phenomenon is the role of the fetus as a sink for maternal lead. Ong et al.[10] have shown that in 25% of their cases, cord blood lead levels exceeded maternal blood lead at delivery,

suggesting that such gradients can exist. In those cases, low maternal blood lead levels may be indicative of increased fetal exposure.

Bellinger et al.[11] also reported an exposure related trend in small for gestational age infants, but no significant association with birth weight was found in their study. Again, almost all of the subjects had blood lead levels below 20 ug/dl.

Taken together, these studies present a qualitatively coherent picture of an association between blood lead levels and reduced birth weight even at blood lead levels well below the current CDC guidelines, and indeed usually at half that level.

Correlation Between Lead and Growth Rates

Two recent prospective studies on children with low blood lead levels have examined the effects of lead on post-natal growth. Shukla et al.[12] followed a cohort of 260 infants from the Cincinnati study to examine growth between ages 3 and 15 months. He found an interactive effect between post-natal and pre-natal exposure. High prenatal exposure suppressed growth, but this effect was reversible if high post-natal exposure was avoided. High pre- and post-natal exposure had the strongest effect. Unfortunately, the term "high" in this context refers to exposure above the median pre-natal exposure of 7.7 ug/dl, a quite low level. In children whose pre-natal exposure was above the median, post-natal increases in blood lead levels were strongly associated with slow growth. The magnitude of this effect was a 2 cm difference in stature at 15 months between those infants who averaged 3 ug/dl during the previous year and those who averaged 15 ug/dl during that period.

Similarly, Lyngbye et al.[13] reported on a population based study of school children in Aarhus, Denmark. Children were classified into high and low groups based on lead concentrations in circumpulpal dentine. The children were measured 3 or 4 times between ages 6 and 10 years. Dietary factors were evaluated and data on maternal smoking during pregnancy obtained, as were socio-economic factors and medical histories. After considering all of these factors, lead remained associated with lower growth in this population, whose mean blood lead level was below 6 ug/dl.

Correlation Between Lead and Gestational Age

As noted above, the Cincinnati study found a significant association between maternal lead exposure and gestational age. In addition, the McMichael et al.[9] study found a significant association between maternal lead levels and pre-term delivery. In the McMichael et al. study, mothers with blood lead levels above 14 ug/dl had more than a four-fold relative risk of premature delivery compared to mothers with blood lead levels below 8 ug/dl.

Lead and Congenital Anomalies

The assessment of a relationship between lead and congenital anomalies is difficult, because the prevalence of malformations is low. Therefore, large sample sizes are required to detect effects. Nonetheless, three studies have looked for an association between lead levels and malformations. Needleman et al.[14] reported that lead was associated with increased relative risk of minor malformations of all kinds, with a relative risk at cord blood lead levels of 15 ug/dl more than twice that for 0.7 ug/dl. This study, with a sample size of over 4,000, had a relatively high power, particularly because all malformations were grouped together. It does lose specificity because of that grouping, making interpretation more difficult. On the other hand, McMichael et al., in their study of 774 subjects, did not find any association with congenital anomalies. Ernhart et al.,[15] studying 185 subjects, also failed to find an effect. It is not clear whether the difference between the studies reflects primarily sample size, or a contrary finding, since the insignificant studies have not published regression coefficients to compare to those of Needleman et al. No definitive conclusion can be drawn under these circumstances.

Metabolic Studies

Correlation Between Lead and Vitamin D Metabolism

Rosen[16] and Mahaffey[17] have reported strong correlations between blood lead levels and circulating levels of 1,25-$(OH)_2$ Vitamin D in children. These correlations are seen across the whole range of blood lead values from 12-120 ug/dl. They appear to result from lead inhibiting the hydroxilation of the 25 to the active 1,25 form of the vitamin. At 35 ug/dl, the suppression of Vitamin D activity reaches levels comparable to those seen in severe kidney dysfunction and several genetic disorders.[18] The consequent disturbance in calcium metabolism that is produced by lead may relate to the growth and developmental effects of lead. Because calcium serves as the second messenger for numerous cellular metabolic processes, these results, as well as Habermann's[19] finding that lead can replace calcium in activating calmodulin, suggest a more widespread disturbance in metabolism in the exposed child.

Correlation Between Lead and Pituitary/Thyroid Function

Two studies have suggested that lead impairs the pituitary-thyroid endocrine system in a manner that may be related to its effect on growth. Stanstead[20] has shown a lead induced impairment of the iodine-concentrating mechanism by lead in rats and in men. The effect was reversible upon injection of thyroid stimulating hormone (TSH), a pituitary hormone, suggesting the effect was mediated by suppression of TSH. More recently, Huseman et al.[21] reported that two lead intoxicated children had decreased TSH release in response to TRH. They

incubated rat pituitary cells, and found that those exposed to lead again showed decreased TSH release in response to TRH. This interference in pituitary/thyroid functioning may well relate to the correlation between lead exposure and growth.

Interaction of Lead with Iron Deficiency

Two recent analyses of the NHANES II data indicate that low level lead exposure can interact with iron deficiency, yielding enhanced effects and effects at lower levels in iron deficient children. Recently, Mahaffey and Annest[22] analyzed the relationship between blood lead levels and free erythrocyte protoporphyrin levels (FEP). They reported greater increases in the proportion of children with elevated FEP levels as blood lead increased, when the children were iron deficient (transferrin saturation below 16%).

Marcus and Schwartz[23] have also analyzed this data, fitting a toxico-kinetic model to the relationship between blood lead and FEP, stratifying on iron status. Their model allows the prediction of the concentration of lead in plasma and body fluid, as opposed to simply in the blood, where over 95% of the lead is bound to the erythrocyte and less toxicologically available. They also report an interactive effect with iron status. In children with transferrin saturation below 14%, the threshold for lead elevating FEP levels is at 12 ug/dl; for high iron children, it did not begin until 23 ug/dl. These results should be compared to Piomelli et al.'s[24] threshold of 17 ug/dl for all children.

These effects are unlikely to be restricted merely to FEP elevations. The relationship between plasma lead and blood lead has been shown to be non-linear.[25] The parameters estimated in Marcus and Schwartz indicate that the plasma levels obtained at a blood lead of 25 ug/dl in children with average iron levels occur at a blood lead of 20 ug/dl in the iron deficient children, and not until 30 ug/dl in children whose transferrin saturation exceeds 31%. Given the large difference in the toxicological availability of erythrocyte and plasma lead, it is likely that heme synthesis is not the only effect of lead that will occur at lower blood lead levels in iron deficient children. Because both lead exposure and iron deficiency are associated with poverty, these children may be doubly at risk.

II. OTHER NEUROLOGICAL EFFECTS OF LEAD

Correlation Between Lead and Hearing

In 1985, Robinson et al.[26] reported a linear increase in the 2 KHz pure tone hearing threshold as blood lead levels increased from 6 to 47 ug/dl. There was no sign of a threshold. To confirm those results, and to examine hearing thresholds at other frequencies, Schwartz and Otto examined the hearing data from the NHANES II study.[27] Lead was positively associated with hearing loss at 500, 1000, 2000, and 4000 Hz. Figure 4 shows the relationship between blood lead levels and

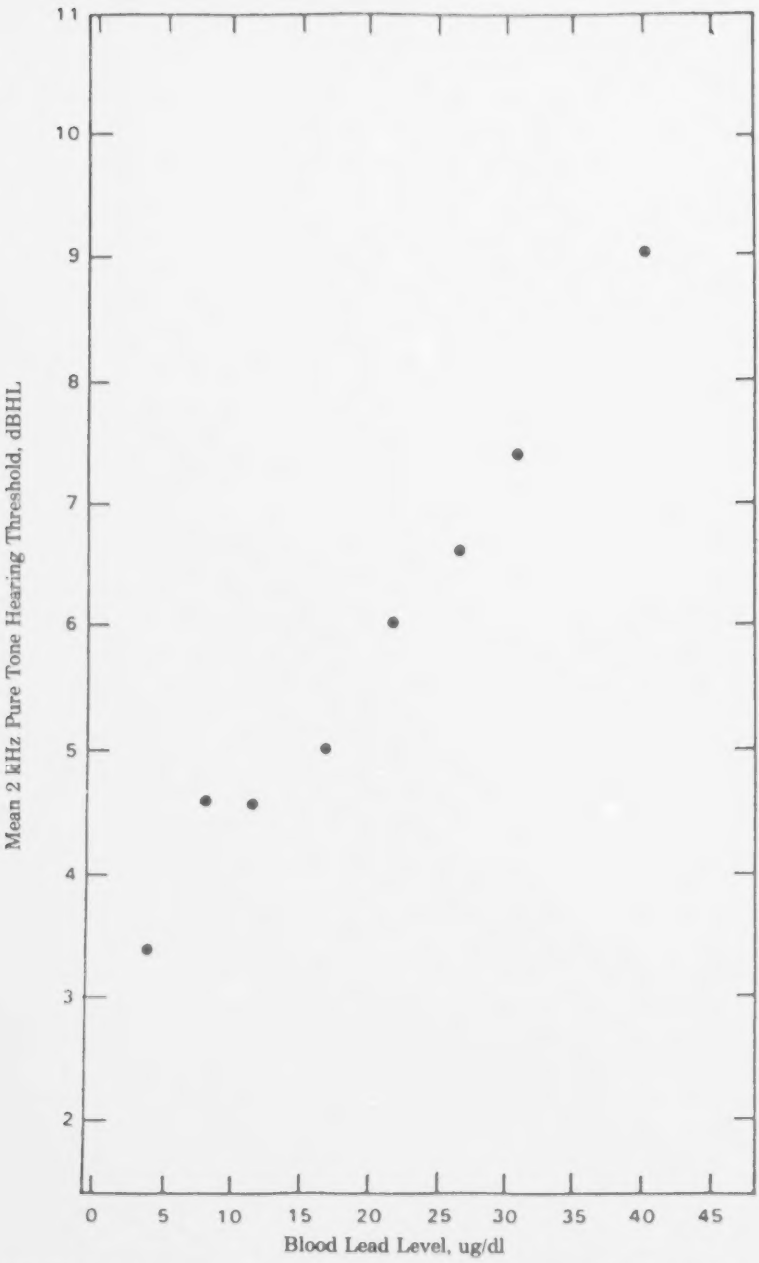

FIGURE 4. Relationship of 2 kHz pure tone hearing thresholds and blood levels in NHANES II subjects aged 14-19 years. Each point represents the mean hearing threshold of all persons in a 5 ug/dl blood lead range.

hearing thresholds for all children aged 6 to 19 in the NHANES II survey. Children with blood lead levels of 25 ug/dl had an average 3 decibel hearing loss compared to children with 5 ug/dl of blood lead.

Hearing loss at higher exposure levels has been reported in occupationally exposed workers. However, Repko and Corum have criticized those studies for inadequate matching by age.

Lead Induced Peripheral Neuropathy

While the peripheral nervous system is generally less sensitive to toxicants than the central nervous system, a recent study in adults showed decreased peripheral nerve conduction velocity at blood lead levels as low as 40 ug/dl.[28] This prompted Schwartz et al.[29] to re-examine an earlier study in children[30] to determine the blood lead level where peripheral neuropathy began. Two forms of analysis were used. In the first they postulated a uniform threshold for all children, and fit two lines to the data on nerve conduction velocity above and below the threshold lead value. The threshold value was varied to find the level with greatest explanatory power. In the second model, they postulated that threshold levels vary among children, following a normal distribution. This produces a logistic shaped dose response curve for the whole population. This curve was then fit to the data.

In both cases they found that blood lead levels of 30 ug/dl and higher were associated with decreased nerve conduction velocity in children. The logistic model indicated that in the more sensitive children, the threshold may be in 20-30 ug/dl range. Again, these levels are lower than those previously thought to cause demyelinization.

Conclusions

The accumulated evidence points to a role of lead in disturbing physical development in the fetus and the child at levels well below those once thought to be safe. The evidence from studies of birth weight, gestational age, malformations, post-natal growth, and post-natal attained stature, when taken together, show a remarkable consistency.

Recent toxicological studies suggest that disturbances in heme biosynthesis and heme related enzymes, in hormonal production, and in the function of calcium in mediating metabolic processes all occur at blood lead levels below 25 ug/dl. Again, the likely causal of these mechanisms in the effects cited above strengthens the conclusions of each separate set of studies. Given the extensive role of calcium in regulating metabolic processes, and the equally ubiquitous role of heme related enzymes, it is possible that further investigations will identify other subtle, but potentially important disturbances caused by low level lead exposure. The finding of peripheral nerve disturbances at lower than expected levels, and of hearing loss as an additional possible effect of lead only adds to this developing concern.

While medical intervention for children below 25 ug/dl is not an option, this is a reflection of the inability of medicine to provide therapy safe enough to make its administration prudent at those levels, not a reflection of lack of toxicity. While this situation is likely to continue for the foreseeable future, environmental intervention to prevent these exposure is clearly needed to protect children, and developing fetuses. Lead poisoning and prevention programs need to incorporate these interventions in the future, and national action to reduce common exposure sources is also needed.

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SUMMARY OF LEAD-BASED PAINT REGULATIONS

Presented by Carolyn Newton

The history of the Lead-Based Paint Regulations goes back to 1971 when the Lead-Based Paint Poisoning Prevention Act (LBPPPA) was passed by Congress with HEW as the lead agency. This act prohibited the use of lead-based paint (LBP) in residential structures constructed or rehabilitated by the Federal government or with Federal assistance. In 1973, Section 302 was added to the LBPPPA, making HUD the lead agency and directing them to establish procedures to eliminate, as far as practicable, LBP poisoning in existing housing constructed before 1950, covered by mortgage insurance or housing assistance payments. At a minimum, these procedures were to eliminate the immediate hazards to children, and notify purchasers and tenants of LBP hazards, symptoms, treatment, and abatement techniques. HUD had discretion to apply these procedures to housing constructed during or after 1950. HUD also was directed to establish and implement procedures to eliminate the hazards of LBP poisoning in federal properties, prior to their sale, if their use was intended for residential habitation.

HUD began its publication of regulatory requirements by issuing 24 CFR Part 35 in 1972 which prohibited the use of LBP in Federal and Federally assisted construction. In 1976 they published regulations implementing Section 302 of the LBPPPA which extended Part 35 to all HUD associated housing, including all HUD financially assisted housing when sold, bought, leased, constructed or rehabilitated. This publication also required notification to purchasers and tenants in pre-1950 HUD housing of LBP hazards; hazardous paint elimination in HUD housing regardless of construction date; and the treatment of any paint categorized as an immediate hazard, i.e., cracking, scaling, chipping, peeling, or loose paint. This regulation did not require testing for lead content, and excluded intact paint.

In 1983, public housing tenants in Washington, D.C. challenged HUD's regulations in the courts through the *Ashton v Pierce* case, asserting that HUD regulations were deficient because they did not treat LBP accessible to children as an immediate hazard, and they did not prescribe sufficient steps to eliminate accessible LBP. The court ruled that HUD had not provided adequate guidance and ordered them to revise their regulations. The courts held that an "immediate hazard" was not limited to paint in a defective condition; that cost and technical considerations could be considered when developing regulations; that the threshold of practicability is reached if reasonably

available techniques for eliminating a hazard exist; and that HUD has broad discretion to determine what paint is an "immediate hazard."

HUD, therefore, revised its general regulations in August, 1986 and January, 1987. The changes included:

1. Directing each HUD Assistant Secretary to prescribe a LBP hazard notice for purchasers and tenants of HUD housing, constructed prior to 1978;
2. Requirements and conditions for the elimination of the LBP hazard in HUD housing to include a 1950 construction cut-off date, mandated removal from defective paint surfaces only, requirements for inspections to identify the problem in a dwelling unit, treatment methods authorized, and authority to supersede the regulations;
3. The requirements to eliminate LBP in federally owned properties prior to use as residences were modified by adding a 1950 construction cut-off date, and adding the same inspection and treatment requirements used in Subpart C;
4. Other program specific regulations were also modified and superseded 24 CFR, Subpart C. These regulations also deal with the hazards of intact (chewable) LBP and address testing and abatement methods. The dates of the new regulations are:

   Public and Indian Housing: August 1, 1986
   Insured Housing and Section 8: January 15 and March 27, 1987
   Rehabilitation Programs: February 17, March 24, and April 9, 1987

There are some common terms utilized in HUD LBP regulations which define the housing and surfaces covered by these regulations. They are:

1. *HUD-associated housing*—Any residential structure that is the subject of an application for mortgage insurance under the National Housing Act or is proposed for the receipt of housing assistance payments under a program administered by the Secretary. For purposes of Subpart A, "HUD-associated housing" also includes any existing residential structure:
   (a) Acquired by the Secretary pursuant to any provision of law which, prior to such acquisition, was insured under the National Housing Act or was subject to a loan under Section 312 of the Housing Act of 1964,
   (b) Sold by the Secretary following any such acquisition and subject to any requirements regarding its use or operation under an agreement with, or condition imposed by, the Secretary, or
   (c) That is currently covered by mortgage insurance or a contract for housing assistance payments.

2. *Residential structure*—Any house, apartment or structure intended for human habitation, including any non-dwelling facility operated by the owner and commonly used by children under seven years of age, such as a child care center.
3. *Applicable surface*—All exterior surfaces of a residential structure, up to five feet from the floor or ground, such as a wall, stairs, deck, porch, railing, window, or doors, which are readily accessible to children under seven years of age, and all interior surfaces of a residential structure.
4. *Chewable surface*—All chewable protruding painted surfaces up to five feet from the floor or ground, which are readily accessible to children under seven years of age, *e.g.*, protruding corners, windowsills and frames, and other protruding woodwork.
5. *Defective paint surface*—Paint on applicable surfaces that is cracking, scaling, chipping, peeling or loose.
6. *Elevated blood lead level or EBL*—Excessive absorption of lead, that is, confirmed concentration of lead in whole blood of 25 ug/dl (micrograms of lead per deciliter of whole blood) or greater.
7. *Lead-based paint surface*—A paint surface, whether or not defective, identified as having a lead content greater than or equal to 1 mg/cm^2 (milligram per square centimeter).

Based upon HUD's practicability analyses, construction cut-off dates vary depending upon the HUD program involved. Generally, there are two cut-off dates for construction which pertain to LBP surfaces. They are, 1978 for defective paint and 1950 for chewable paint.

Inspection and testing criteria were also updated. Inspection for defective paint can be done visually. In most HUD programs, inspection for chewable paint must be done if elevated blood levels are present, must be done using an XRF analyzer, and must be used for random sampling. Laboratory chemical analysis may be authorized by HUD in certain cases. There is a need for an acceptable standard to support the chemical analysis however.

There are three types of Abatement Methods which are acceptable to HUD. They are:

1. Covering—*e.g.*, wallboard, fiberglass cloth barrier, wallpaper which is permanently attached.
2. Removal—*e.g.*, scraping, heat treatment.
3. Replacement

Machine sanding, propane torches, washing and repainting are prohibited methods of abatement.

Issues such as tenant protection, disposal of LBP debris, record-keeping requirements, determinations of comparability, monitoring, enforcement, and funding were discussed in the workshops.

LEAD IN WATER, GASOLINE AND SOIL: THE EPA PERSPECTIVE

Ronnie Levin, M.S.

Lead is a dull gray metal with many commercial applications. It is so intimately associated with the transport of water that the very word *plumbing* comes from the Latin word for the metal. Lead is used in paint, in gasoline, in automobile batteries, in glass and crystal manufacturing, in bullets, to stabilize PVC plastic pipes, and in stained glass windows. It was used for centuries in the manufacture of pewter and in pencils. Lead is used to produce brightly colored pottery enamels. It is a key economic commodity. Its usefulness results from its malleability, durability, its tendency to chalk, and the fact that it doesn't rust. It is also toxic to humans and many other species.

EPA's Current Regulatory Agenda for Lead

The environment is an intricate interdependent and dynamic organism. It is fragile, it is complicated, it is alive. The lead that has been introduced into the environment will circulate through it forever. Over the millennia, lead has become a multi-media pollutant that resides in all environmental compartments and constitutes a human exposure pathway in each.

EPA regulates lead and all other environmental contaminants by media or source. That is, we regulate air pollutants separately from drinking water contaminants, pesticides separately from hazardous wastes, etc. The reason for this derives from our enabling and authorizing statutes, each of which—Clean Air Act, Safe Drinking Water Act, and the like—was written by a separate Congressional committee. This decompartmentalized approach is particularly unsatisfactory for multi-media pollutants like lead.

To compensate for the inherent weakness of such an approach, EPA has tried to maintain a consistent agency-wide perspective on lead. This is not easy.

Lead in Drinking Water

Many studies have shown a strong relation between the amount of lead in people's water and the levels of lead in their blood.[1]

Drinking water is regulated under the Safe Drinking Water Act, passed initially in the mid-1970s and amended in 1986. In November 1985, EPA proposed to reduce the levels of lead in drinking water. The mandatory limit was 50 parts per billion; the proposal was to reduce the health-based goal (not the enforceable limit) to 20 ppb. In 1986,

Congress established statutory deadlines for EPA to issue final standards for about 80 contaminants, including lead. Since the original 1985 proposed reduction and since the 1986 Congressional mandate, EPA has been in "regulatory mode." That means, we're thinking.

We're thinking about what the health-based goal should be; obviously, from a health perspective it should be zero. But if zero lead in drinking water isn't feasible, then that standard is meaningless in the real world. But how can EPA establish any other health-based goal?

We're also thinking about what the mandatory standard for lead in drinking water should be. Lead generally contaminates drinking water, not in the source water, but from the corrosive action of the water upon the materials of the public and private plumbing systems. The largest contribution is from the lead solder that joins copper pipes in most of the homes in this country. While EPA probably can't do much about the plumbing in your house, we can require that the public water system supply water that is as non-corrosive as possible. This will keep the water from corroding the lead out of the plumbing materials and into your water.

Fortunately, in general, reducing the corrosivity of water is neither particularly difficult nor expensive. The main parameters that influence corrosivity are pH, alkalinity, and hardness. Raising pH or adding lime to the water can be done at a cost of well under $1 per person per year, including capital, and many cities, including Boston, Seattle, Bennington VT, and Manchester NH, are already treating their water for corrosivity.

At present, it looks like EPA will propose not an enforceable limit for lead in drinking water at your home, but guidelines for making water non-corrosive. While that's a little like requiring disinfection and not requiring that bacteriological standards be met, EPA's lawyers are saying that is what we must do. Current estimates are that both the health-based goal and the mandatory standards (whatever they turn out to be) will be proposed in Spring 1988.

Another part of the problem of lead contamination of drinking water was addressed by the 1986 amendments to the Safe Drinking Water Act. An amendment banned the future use of materials containing lead in public water supplies and residences connected to them. Enforcement of this ban, however, was turned over to the states and not to EPA.

Lead in Air

The Clean Air Act, passed originally in 1970 and amended several times since then, requires EPA to set National Ambient Air Quality Standards (NAAQS) for a variety of air pollutants, including lead. While these standards should be set to protect the most sensitive population with an adequate margin of safety, lead contamination is so pervasive that even under the most stringent standards thousands of children will still be lead-poisoned every year.

The Clean Air Act calls upon EPA to revise the air standards every 5 years, but EPA has been unable to keep that schedule. An NAAQS for lead was last set in 1978 and EPA is currently reviewing this standard. A new standard may be proposed next year. The combustion of leaded gasoline was by far the greatest source of ambient air lead (and indeed the major source of environmental lead contamination altogether). Since the phasedown in the allowable limit for lead in gasoline, stationary sources such as smelters have become relatively more important; leaded gasoline is still a significant contributor, however.

Interestingly, it is the process established to regulate air lead that most governs EPA's multi-media lead policy. I think this is primarily historical—EPA's air and research offices began to address lead contamination together a decade ago. The drinking water office has not been involved, although arguably it should have joined this effort many years ago. We are just now learning about lead-contaminated soil, which is being addressed currently under the Superfund program.

Lead in Gasoline

Many studies[2] have shown quantitatively the close association between the combustion of leaded gasoline and blood lead levels. To address the health and environmental hazards posed by leaded gasoline, EPA has regulated it since 1973 to meet 2 distinct goals: assure the availability of unleaded gasoline to those vehicles with pollution-control devices (catalysts) that are rendered ineffective by leaded fuel and reduce the adverse health effects associated with exposure to lead. EPA's most recent phasedown reduced the allowable limit for lead to 0.10 grams per leaded gallon. At the same time that EPA "went final" on this latest phasedown, the agency also proposed a ban on the sale of all leaded gasoline. That proposal is still on the books, although there is no indication that it will be finalized.

Leaded gasoline, with some 100 billion gallons sold annually for many years, stands tallest among the sources of lead pollution. In fact, it dwarfs the other contenders. Lead from gasoline can fall into many environmental compartments: what is not inhaled directly can settle as dust on your dishes or the spinach waiting to be picked, it can enter drinking water reservoirs or contaminate the soil in a playground. The possibilities are endless. During the 1970s, leaded gasoline contributed an average of about 8 ug/dl to blood lead. The phasedowns over the past decade have resulted in a 50% average reduction in blood leads in this country. Unfortunately, however, past use will continue to affect us for a very long time as current ambient air settles into dust.

Lead in Soil

Lead contaminates soil from 2 primary sources: deposition from ambient air, especially emissions from leaded gasoline and stationary sources, and from the intentional and unintentional flaking of leaded

paint. These 2 sources also contribute significantly to dust lead, both within the house and outside. Lead in soil and dust is another major route of exposure to lead.[3] The relationship is strongest for toddlers and young children, due to mouthing and play activities.[4]

Lead-contaminated soil has long been associated with areas adjacent to smelters and with hazardous waste sites. More recently, however, attention has moved from those (usually relatively small) areas likely to produce high exposure to sites in urban areas with sufficient contamination to produce chronic lower level toxicity. This is the situation in many urban areas, including Boston. A pilot program, funded through Superfund, is now evaluating the efficacy of reducing blood lead levels through the removal of contaminated soil. A total of $18 million was appropriated for this investigation; Boston was chosen as the first pilot.

Several areas in Boston were identified as most likely to have highly contaminated soil. Criteria for the identification of such areas included demographic, environmental and socio-economic conditions, although conditions vary greatly street to street and even house to house. Soil lead concentrations at selected homes ranged from 180 ppm to 11,400 ppm, with a mean of 1,870 ppm. House dust levels were higher.

What was particularly appealing about this lead exposure reduction strategy was that soil removal is much cheaper than paint removal. At least at the levels originally perceived. The initial soil tests showed that lead contamination only occurred in the top 3 inches of soil; a conservative mitigation strategy to remove 6 inches of soil from a 6 foot ring around the house was estimated to cost $1,500 per house. Subsequent testing showed elevated lead levels at least 8-10 inches down and across the entire yard; current soil removal efforts are estimated to cost over $5,000 per house. This has more than tripled the cost of just the mitigation component of the study. The survey and analytical parts are now also projected to cost more than originally anticipated. As of about a month ago, the status of this pilot was in doubt.

EPA's Goal: Get the Lead Out! Provided . . .

EPA's current goal is to reduce lead exposure to the maximum possible. The question is, What is possible?

In policy decisions relating to setting NAAQS under the Clean Air Act, EPA has committed itself to actions that will bring 99.5% of the children in the country to blood lead levels under 15 ug/dl by 1992. To accomplish this goal, mean blood lead levels for children will have to be about 5-6 ug/dl. [Addendum: Since December, 1987, EPA has reduced its blood lead level of concern to 10 ug/dl. The goal of both the NAAQS and the proposed standard for lead in drinking water is now to keep 99.5% of U.S. children below 10 ug/dl. This will require a mean blood lead level of slightly under 5 ug/dl.]

EPA has statutory authority to regulate lead in the air, soil, and water. To achieve its 1992 health goal, EPA will have to regulate lead in several media. Two regulatory actions, mentioned above, are already underway. First, the agency proposed a total ban on leaded gasoline 3 years ago but has never promulgated that ban. Second, EPA proposed to lower lead in drinking water 2 years ago but has not yet promulgated that rule either. In addition, studies relating to soil contamination and emissions from stationary sources are being conducted. EPA must choose from among alternative strategies to accomplish a single goal, in this case, reducing exposure to lead.

Competing for Resources: the case for cost-benefit analysis

An Executive Order was isued in 1981 that required all federal agencies to estimate the costs and benefits that will result from any anticipated action. Ideally, these cost-benefit analyses provide an immediate and obvious decision-making tool. For instance, to evaluate how low to set an air level for lead, one need only compute the point at which the marginal cost exceeds the marginal benefit. Then you stop one step before that point.

Cost-benefit analysis enables decision-makers to decide how to allocate scarce or limited resources. Resources for reducing lead exposure and for medical services are limited. In the case of lead-paint mitigation versus lead-soil removal, for instance, one compares the benefits and costs of one method against the other. The bigger gain wins.

A problem, however, is that cost-benefit analysis makes people nervous. Health professionals and environmentalists are afraid that the costs of protection will outweigh the benefits. Industry people only support such analyses when they show that the costs are too high.

Analytically, the greatest limitation of the method is that the costs are much easier to calculate than the benefits. Often, we don't even know how to calculate the benefits; we cannot estimate how many trout will spawn or how many trees will grow better leaves or how many fewer cases of respiratory disease will be avoided. Where we can estimate some of these effects (which is usually a small subset of the total benefits that will occur) we cannot translate those gains into dollars. We are then left, at best, comparing apples and oranges.

Ultimately then, decisions are based upon *perceived* costs and benefits. Because the costs are always more easily quantified than the benefits, I believe that in these circumstances the benefits tend to be seriously underestimated, particularly when there are health effects involved. Better results will occur as our ability to quantify benefits improves.

Lead is a good case for cost-benefit analysis. We know more about the routes and sources of lead exposure and resulting health effects than about probably any other pollutant. We also know how much it will cost to reduce those exposures. In general, the benefits of reducing lead exposure greatly outweigh the costs. Table 1 contains some of the

yearly health benefits of the two regulations EPA is now considering. In both cases, the annual health benefits are more than double the annual costs, and we know that the benefits we've estimated are incomplete. In both case, there are also other benefits of reducing lead besides the health gains. The total annual benefits of each action are about three times greater than the annual costs.

When Other Interests Get in the Way . . .

Obviously, many factors are considered in making decisions. For instance, both the potential ban on the sale of leaded gasoline and the reduction in the allowable level for lead in drinking water are stymied at present by special interest groups and legal and technological concerns. These include farmers, antique car owners, the petroleum refineries and public drinking water officials. Frankly, you are also a special interest group. The political process in this country is open to everyone.

Making your position known is not only done with money. Letters —personal or from your professional affiliations—are effective attention-getters. One letter will neither reverse a decision nor force one, but many letters carry a clear message to decision-makers: "we care about this issue and we are watching what you do."

EPA's decisions about the levels of lead in air, drinking water, and soil will directly affect how many children you have to treat. Make sure that we know what you think.

Conclusions

From EPA's perspective, regulating lead is a good idea. Reducing exposure to lead is probably the most important public health action the agency will take this decade. Easily half of the U.S. population is at blood lead levels at which detectable health effects have been indicated. It is the only disease for which we discuss what percentages of the population are at risk. And in this case, we have sufficient knowledge to be able to quantify the benefits. As can be seen in the summary table on the next page, the yearly benefits, both in dollars and in people, are enormous.

Summary of Monetized and Non-monetized Annual Health Benefits of EPA's Proposed Ban on Leaded Gasoline and Reduction in Lead in Drinking Water

Effect	*Gasoline Ban*		*Drinking Water*	
	# of people	*$millions*	*# of people*	*$millions*
CHILDREN (reductions in numbers of children at risk)				
requiring medical treatment	7,000	$6.7	29,000	$27.6
loss of IQ points				
1-2 points	83,000	$86.3	230,000	$239.2
4 points	2,000	$5.2	11,000	$28.6
5 points	50	$0.1	100	$0.3
requiring compensatory education	7,000	$19.6	29,000	$81.2
growth decrement	26,000	NV	82,000	NV
fetuses at risk	NC	NV	680,000	NV
ADULT MALES (reductions in numbers of males at risk)				
cases hypertension (males, aged 40-59)	123,000	$30.8	130,000	$32.5
myocardial infarcs (white males, 40-59)	200	$13.0	240	$15.6
strokes (white males, 40-59)	70	$3.4	80	$3.8
deaths (white males, 40-59)	200	$200.0	240	$240.0
TOTALS ($millions)		$365.1		$668.8

Notes: NV=not valued monetarily; NC=not calculated

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REGIONAL COLLABORATION: THE NECCLPP EXPERIENCE

Amy Zimmerman, M.P.H., R.D.
Martha M. Turner, R.N., B.S.N.
Phyllis M. Madigan, B.A.

The theme of this workshop was to explore ways in which to develop regional networks and collaborative efforts to fight lead poisoning, and to demonstrate the benefits of such efforts utilizing the experiences of The New England Consortium of Childhood Lead Poisoning Programs (NECCLPP). A brief history of the Consortium's development was presented. Participants were then divided into groups and given additional background information including the status of lead poisoning activities at the time of NECCLPP's inception and NECCLPP's original ten objectives. The groups were then asked to identify how they would pursue achieving the stated objectives (type of administration and organizational structure to implement, type of activities to sponsor, ways to evaluate their efforts and ways to support activities when funding no longer exists).

The groups presented their ideas to each other after which an overview of NECCLPP was presented which identified it as one model which has enhanced collaborative efforts on a regional basis. Additionally, to demonstrate the type of benefits which have resulted, two examples were discussed in detail. They included the evolution of New Hampshire's Childhood Lead Poisoning Prevention Program along with the accomplishments of NECCLPP's Laboratory Task Force. included below is an overview of NECCLPP (history, goals, accomplishments) along with two detailed accounts of how NECCLPP has served an individual state as well as laboratory services through the region.

An Overview of the NECCLPP Services

History

The New England Consortium of Childhood Lead Poisoning Programs (NECCLPP) is a regional model which has encouraged and provided for the strengthening of childhood lead poisoning programs in New England. NECCLPP evolved as a result of the administration and fiscal changes precipitated by the Omnibus Budget Reconciliation Act of 1981 (replacement of categorical funds with the MCH block grant) which presented a serious challenge to the effective and efficient management of childhood lead poisoning programs in New England. The simultaneous identification of an expanded population of children

at risk intensified the need for an innovative management strategy for childhood lead poisoning prevention services. Rhode Island, with the endorsement of the five other New England states, submitted a proposal to strengthen and sustain childhood lead poisoning prevention programs in New England. In 1982 the Division of Maternal and Child Health, Department of Health and Human Services, awarded the Rhode Island Department of Health funds to support the New England Consortium of Childhood Lead Poisoning Programs.

The Purpose

The primary goal of NECCLPP is assist states in the planning, management, and evaluation of childhood lead poisoning prevention programs. The Consortium coordinates, advises and facilitates regional efforts to reduce lead exposure and lead toxicity among children. NECCLPP is actively concerned with all aspects of lead poisoning prevention programs including needs assessments, state and local management of resources, screening strategies, clinical care standards, legal and enforcement bases, environmental strategies, new technologies, laboratory resources, public education, data systems, staff training, funding sources, etc. The Consortium also provides a mechanism by which newly identified national, regional and local lead related issues can be addressed. In order to accomplish the above goal, NECCLPP established and has been working towards 10 objectives which are listed below:

1. To assist state MCH authorities and other interested parties in the assessment of need for targeted childhood lead poisoning prevention activities.
2. To identify needs for training and technical assistance among childhood lead poisoning prevention programs in the region.
3. To develop an inventory of technical assistance resources within the region and nationally.
4. To design, coordinate, and conduct training programs for management and field personnel responsible for childhood lead poisoning prevention programs.
5. To arrange technical assistance as appropriate for states and programs, drawing on existing staff resources within the region so much as possible.
6. To develop guidelines and protocols for needs assessment, program planning, management, evaluation, and quality assurance in childhood lead poisoning prevention programs.
7. To serve as liaison between childhood lead poisoning prevention programs and relevant research and evaluation initiatives in the region.
8. To foster the use of effective new technologies and strategies for lead poisoning prevention.

9. To encourage and assist in the collection and analysis of common databases and program evaluations of childhood lead poisoning prevention activities.
10. To encourage development of regional resources in those areas where multi-state collaboration will foster program effectiveness and/or reduce costs (e.g., in training, laboratory work, dissemination of new technology, etc.).

Organizational Structure

NECCLPP is organized to execute its mission through a six state Executive Committee, four Task Forces (Data, Laboratory, Medical Education, and Community Education) and a full time coordinator. The Executive Committee, consisting of the six states' Maternal and Child Health Directors or their designees, are ultimately responsible for policy making and program planning decisions, identifying priorities, and establishing the Consortium's goals and objectives. The four task forces address individual matters of concern in their respective areas. This enhances regional networking among all levels of professionals. Additionally, this process identifies and utilizes the expertise of many dedicated professionals throughout the region. Participation in these committees and the strong leadership at the state level have assisted NECCLPP in developing the sustaining childhood lead poisoning prevention programs. Lastly, the coordinator oversees the day-to-day activities of the Consortium and assumes responsibility for the administration of Consortium activities. This overall organizational structure has allowed NECCLPP to assist in strengthening components of existing programs, developing new regional resources and developing a regional network of collegial communication to sustain interstate collaboration.

Activities

The emphasis of NECCLPP's initial project period focused on the development of the Consortium as a working unit, the definition of individual states needs and resources, and the initiation of strategies to provide technical assistance and training. A number of Special Projects in individual states have been supported by NECCLPP funds. These projects have allowed NECCLPP not only to strengthen individual components of existing childhood lead poisoning programs, but also to promote and develop new resources in areas where children are determined to be at risk and for whom lead poisoning prevention services did not exist. NECCLPP also sponsors a variety of conferences and training workshops, provides on-site consultation and disseminates information via newsletters. Through the task forces, educational materials have been produced, laboratory studies have been conducted, clinical symposiums and grand rounds have been conducted, regional quarterly report forms have been developed and data

has been collected, a two state evaluation study has been conducted, and a survey of MCH agencies (WIC, EPSDT, Day Care, Head Start) was conducted to define common service population and promote lead poisoning activities. In addition, NECCLPP coordinates the loaning of equipment regionally.

Listed below are several specific activities which have resulted as outcomes of NECCLPP.

Special Projects have included:

- a one-year lead poisoning prevention project in the state of New Hampshire which allowed them to implement and maintain a state-wide lead poisoning prevention program. NECCLPP provided additional support to reach two high risk populations: Southeast Asian children and children of parents occupationally exposed;
- a one-year prevalence study to determine the extent of lead toxicity and iron deficiency in Vermont and the development of a deleaders manual for contractors engaged in renovation;
- a regional lead-in-soil project to determine the costs and benefits associated with obtaining soil analysis for lead during the environmental inspections and to investigate the extent of lead contaminated soil in Portland, Maine;
- a needs assessment conducted for the State of Maine;
- a needs assessment in the State of Connecticut.

Major Symposiums (for which some have proceedings) included:

- Combating Childhood Lead Poisoning (1983)
- Childhood Lead Poisoning in the 1980's (1984)
- Environmental Lead Exposure—A Hazard to Young Children (1984)
- Clinical Management of Children with Undue Lead Absorption (1985)
- Case Management of Childhood Lead Poisoning, A Program Challenge of the 80's (1986)
- Leading us Away from Lead! Whose Responsibility? (1987)

Educational Materials (produced or being produced):

- Slide show suitable for regional use
- Nutrition brochure
- Renovation brochure
- Chelation therapy brochure
- Lead poisoning prevention poster

Reports and Special Studies:

- New England Public Health Laboratory Testing Services (pilot study of lowered EP threshold conducted in 1985)
- Quantitative analysis of the Public Health Laboratory testing services for NECCLPP (a cost analysis study using workload time units and index, conducted in 1986)
- 1985 NECCLPP Evaluation Study
- A Comparison of Selected Characteristics of the Childhood Lead Poisoning Prevention Programs of Massachusetts and Rhode Island (two state evaluation studies conducted in 1986)
- Quarterly report analysis
- Lead in Soil: A New England Consortium of Childhood Lead Poisoning Program Response to Recent Initiatives
- An Analysis of the Prevalence of Lead Poisoning in Rhode Island's Southeast Asian Population.

Miscellaneous Activities

- Grand rounds at 12 pediatric institutions throughout the region (after surveying hospitals)
- Quarterly newsletters
- Development of data quality report form
- Responses to CDC, EPA and HUD respectively regarding policy statements which impact on New England Programs

As evident, NECCLPP facilitates networking throughout the region; enhances regional communication by addressing a critical environmental public health problem; promotes efforts to facilitate collaborative regional efforts to determine cost effective and timely methodologies utilized in providing lead poisoning prevention services (laboratory services, screening environmental follow-up etc.); provides education and training, and reviews, responds to and develops public health policy regarding lead poisoning prevention services.

New Issues

This is an exciting time in the field of lead poisoning prevention nationally, regionally and locally. The Environmental Protection Agency (EPA) has approved a total of 15 million dollars from the Superfund to investigate the relationship between lead contaminated soil and childhood lead poisoning; Boston has been chosen as the first of three national pilot sites to receive 5 million dollars to conduct such a demonstration project. The Department of Housing and Urban Development has promulgated new regulations around the issue of lead which will place an increased demand on Childhood Lead Poisoning Prevention Programs. New studies are reflecting serious adverse pregnancy outcomes (including low birth weight and developmental

delays) associated with low level prenatal exposure. A Massachusetts Legislative Commission was convened to conduct an investigation of the adequacy of lead poisoning prevention efforts; legislation based on the committee's findings has passed. Connecticut has received some state funding ($50,000) and has submitted a budget option for 1.2 million dollars to be allocated for lead poisoning prevention services. The New Hampshire Department of Health and Welfare submitted lead legislation for the first time to obtain changes in the law and to request funding. Lastly, the State of Maine has shown new interest in addressing lead poisoning prevention efforts.

A number of the above issues (prenatal toxicity, lead in soil and dust, HUD, lower CDC guidelines) will impact upon the childhood lead poisoning programs at all levels (local, state, regional and national) and need to be more fully addressed.

Outlined below are the specific issues which NECCLP has seen evolve as priorities and which merit future attention:

- The extent of lead contaminated soil (and dust) and EPA's Superfund activities;
- The effect of low level lead exposure on pregnant women and their fetuses;
- The impact of the New Housing and Urban Development Guidelines;
- The increase in the demand for screening laboratory and environmental services as a result of the lowered CDC guidelines, HUD's regulations and increased public awareness;
- The need for increased collaboration between MCH, environmental and housing agencies.

The Future of NECCLPP

NECCLPP has been funded as a Special Project of Regional and National Significance (SPRANS) by the Bureau of Maternal and Child Health, Department of Health and Human Services, since 1982. 1988 marks the end of NECCLPP's project period.

As a result of NECCLPP's successes in coordinating and promoting collaborative approaches to prevent childhood lead poisoning within New England, there is a strong desire among the states to continue these efforts. NECCLPP is working to develop a strategy which will enable coordinated regional lead poisoning prevention activities to be maintained. Strategies being investigated include shifting the coordination of regional activities to an already existing regional environmental public health center, pursuing other sources of funding such as private foundations and linking with other grant funded regional projects.

NECCLPP Laboratory Task Force

Origin

The Laboratory Task Force was formed as a result of the first regional lead conference sponsored by NECCLPP, held in Merrimack, New Hampshire, in October of 1983. At a workshop on Laboratory Management, representatives of the Connecticut and Maine Public Health Laboratories presented reports of on-going laboratory activities in their respective states. During the discussion period that followed, it became apparent that laboratories are key elements in the battle against lead poisoning and not just merely service organizations. It was also apparent that there were many common laboratory concerns and other issues relative to lead detection, specific to laboratories, which could be of benefit to public health laboratories and other program components if these experiences were shared at open forums.

Membership

Membership in the Laboratory Task Force was opened to those involved in the day-to-day operation of public health laboratories providing lead and EP testing services. Each of the six New England States is represented. The first meeting was held in Providence, Rhode Island, on January 19, 1984.

Goal of Task Force

The major goal of the Task Force was to develop strategies for regional collaboration resulting in effective and efficient laboratory technologies.

Major Areas of Interest Included the Following:

- Cost methodologies
- Implications of lowering lead and EP threshold levels
- Quality control of analytical technologies
- Role of computers in monitoring laboratory results and program reports
- Environmental sample analysis
- Uniform units for reporting test results

Major Accomplishments of Laboratory Task Force

- Coordination and participation in pilot studies from February to December, 1984, pertaining to lowered threshold levels for lead and EP in advance of the CDC Statement of January, 1985, as a means of forecasting resources necessary to implement the 1985 guidelines.
- Documentation of the New England collaborative experience

describing individual state laboratory lead and EP testing services and resources available in the New England area. The report titled *New England Public Health Laboratory Lead Testing Services* (June 1985) also includes a review of testing methodologies, data capabilities, provider education, environmental testing services, cost methodologies, quality control and results of pilot studies performed in 1984.

- Participation in a collaborative uniform cost analysis to determine costs for lead and EP testing in the New England area. This project was a joint effort by the NECCLPP Laboratory Task Force and the Centers for Disease Control, Training and Laboratory Program Office, Division of Assessment and Management Consultation, Atlanta, Georgia. Participation in this study involved the following:
  - Preparation and review of organizational structures
  - Inventory of all equipment
  - Preparation of annual statistical reports for July 1, 1985, through June 30, 1986
  - Assessment of budget and personnel accounts
  - Documentation of all testing procedures step by step
  - Performance of time studies for each procedure performed to develop work time units
  - Presentation of a training session on cost accounting and time standards by CDC personnel to 12 lead testing laboratory supervisors and chief chemists from New England Laboratories and to 5 representatives of EPA from Boston, Atlanta, and Washington, DC. This session was held in September of 1986 at the Massachusetts Department of Public Health Laboratory.
- Determination of the workload index for individual laboratories as a management tool in determining productivity.
- Documentation of cost study in a report, *New England Public Health Lead Testing Laboratories, Collaborative Cost Analysis and Comparative Management Data*, November, 1987. This report includes the following:
  - A breakdown by state of lead laboratory personnel costs
  - Lead laboratory cost accounts
  - Worktime unit cost computations
  - Test registers showing the actual cost per test by method utilized in each individual laboratory
  - Workload index using full time equivalents to establish productivity levels
  - The Narrative portion from the previous report of June, 1985
  - An example of time studies used in the cost analysis
- Recommendations of the task force appear elsewhere and will be included in the document at a later date.

Benefits of Participating in the Laboratory Task Force

One of the major benefits has been establishing and maintaining open lines of communication among laboratories and programs involved in lead testing in the New England area. Participation in the task force has resulted in a mutual understanding of problems and a mutual sharing of information which has lead to the development of special projects within the groups. Problems facing laboratories have been discussed freely among the group and solutions have been derived at by a team approach lessening some of the individual frustration of the day-to-day laboratory operation while increasing enthusiasm and motivation. Overall the cooperative collaborative atmosphere has resulted in many shared experiences including a sharing of resources, talent and technologies. A total of twelve meetings have been held since January of 1984, each held at a different site in New England so that members of the task force could meet laboratorians from neighboring states while visiting each public health facility to view methodologies and instrumentation first hand. Several laboratories have served as alternative testing sites for neighboring states when instrument malfunctions occurred.

Future of Laboratory Task Force

Members have expressed an interest in keeping the group together. At present, members keep in touch by phone, mail and by attending conferences. They continue to share experiences, new ideas and methodologies. Unfortunately the mechanism for funding travel to attend future task force meetings is lacking at this time.

WORKSHOPS

NEW HAMPSHIRE'S EXPERIENCE WITH THE REGIONAL MODEL

Martha M. Turner, R.N., B.S.N.

To appreciate what has happened in New Hampshire over the past four years, it's helpful to know a little about our history in lead poisoning prevention. Like our two northern New England neighbors, historically an attitude of disbelief has prevailed among professionals and the public alike that: "Lead poisoning doesn't happen in lovely, rural New Hampshire." It has long been perceived here, as in other parts of the country, that lead poisoning is a problem of the crowded inner city, not New Hampshire; Boston, if anywhere in New England. Yet we live in a state with thousands of wooden, clapboard dwellings covered with leaded paint.

Before 1983, there was no statewide screening program in place in New Hampshire. Sporadic screening occurred in some Well Child Clinics and for some children participating in the Child Health Assurance Program (CHAP) under Medicaid. And if an elevation was found, there was no system in place to insure that a confirmatory venous sample was taken or that an environmental inspection, much less abatement or deleading took place.

In 1980, a committee was convened to determine if there was enough of a problem with lead poisoning in New Hampshire to continue using MCH funds for screening. In 1981, a report was issued by the committee which reported their findings. The group looked at approximately 500 screenings done in Manchester, some on children participating in the local WIC (Women, Infants and Children nutritional supplement program), but the majority from CHAP participants. They found a 1% unconfirmed positive rate in these nearly 500 screenings. They concluded that it was not cost effective to do screenings and therefore there was no basis to continue to require lead screening services for CHAP participants or in the Well Child Clinics.

It is interesting to note that the State's Public Health Lab reported a 5.4% positive rate in 225 screening tests in a one month period during the time that the committee was gathering and considering the Manchester information.

But in spite of the 1981 report, concern about lead poisoning persisted and was pursued by a number of local and state public health

professionals. This concern ultimately expressed itself in the form of a grant proposal to NECCLPP in 1983 by the Division of Public Health Services to start a Lead Screening Program.

The grant from NECCLPP provided for the period of one year:

- $37,000 for personnel and equipment;
- training for the new coordinator of the program, utilizing the Consortium's and other states' resources;
- consultation in the design, goals, and objectives of the program;
- training for pediatric consultants for the program; and
- assistance in developing educational materials.

The grant that was awarded had as its primary intent to initiate a statewide screening program. The project plan was to:

- initiate screening in all the MCH funded Well Child Clinics. These clinics sent capillary samples to the State Public Health Lab;
- have two clinic sites using hematofluorometers on site. Originally the intent was to compare a rural site and an urban clinic site. Six months of screening at the rural site identified only one class Ia child and no blood lead elevations therefore it was determined that the hematofluorometer would be more appropriately utilized at another urban site;
- conduct a door-to-door project in one New Hampshire city;
- develop a tracking system to insure appropriate follow-up of elevated screening tests; and
- develop protocols for screening and management.

In order to institute such a project, a flurry of activity took place to provide the necessary training and education, and to equip personnel to do the screenings and necessary follow up. Training sessions were held for Well Child Clinic staff in the use of the capillary tubes for the collection of samples as well as to provide background information and education on the problem of childhood lead poisoning and the necessity of screening and follow up. Training was also necessary for the staff operating the hematofluorometers at the three sites. Educational workshops were provided as well to physicians, health officers, day care licensing personnel and CHAP personnel.

A key component to the program's initial and ongoing success was our network of medical consultants. All pediatricians in the state were invited to express interest in serving as consultants to the developing program. Of the responding physicians, 11 were selected to represent all areas of the state. After educational sessions with John Graef, M.D. of Children's Hospital Medical Center in Boston, this group developed the initial medical protocols and was available for consultation to other physicians. NECCLPP provided additional expertise by arranging for preceptorships with John Graef, M.D., at Children's Hospital for a number of our consultants. This group is now composed of 4 key pediatricians.

Because of this initial "boost" from NECCLPP, New Hampshire now has more than a screening program. we've developed into a Lead Poisoning Prevention Program.

What have we found to date?:

	# Screened	# Confirmed Positive	% Positive
FY 1984 (6 mos.)	2000	0	
FY 1985	5000	24	.5%
FY 1986	6500	48	.7%
FY 1986	7800	38	.5%
FY 1988 (as of 10/31/87)		27	

While the NHANES II data would lead us to expect a rate of 1.2% in a predominantly white rural population like New Hampshire, these numbers don't differ greatly from Massachusetts which reported a .7% positive rate last year.

One of the advantages to the regional concept is not having to reinvent the wheel and instead, to utilize what others have already developed. Our manual data collection system that first year was designed with much assistance from two other states. Since then, our Automated Data Management System has been developed and continues to be refined.

Another vital component of any Lead Poisoning Prevention Program, our State Public Health Lab, has remained supportive and interested. The lab had been doing blood leads for years; first direct lead levels on samples and then using the hematofluorometer when the technology became available. A level of support was already in place when the screening project began. They have actively participated in NECCLPP's Laboratory Task Force. Our ongoing relationship and exceptional level of communication have been facilitated by regularly scheduled meetings to discuss issues of mutual concern as well as frequent informal contact.

The Environmental Component of the program has presented the greatest challenge in the program's development. Doing home inspections for lead paint was a new and onerous responsibility for the designated department. The two XRF's which the department owned (used primarily by one city Health Department) were in disrepair, and funding allocations were unavailable for source replacements or equipment maintenance nor was there sufficient personnel time available. To further complicate the situation, the state's Lead Law was not very clear, no rules were in place, and the law was understood to be unenforceable.

With an investment of time and energy into developing this component of the program and with new personnel in the department who have an interest in lead poisoning, support has blossomed. The en-

vironmental staff have developed an environmental questionnaire; have initiated discussions about developing a system for re-inspection of the homes of lead poisoned children; are taking a large part of the responsibility for working with local health officers and are willing to participate in and provide training related to lead poisoning. Again, the Lead Program's relationship to this department is facilitated by means of frequent formal and informal communication.

In managing cases of lead poisoning, the most critical element of all, environmental management, has posed the greatest challenge—and the most frequent obstacles as well. Health officers in the cities and towns are frequently unfamiliar with the Lead Law and difficulties abound in enforcing the law. Landlords are seldom willing to do the necessary abatement work due to high cost and questionable return on their investment, especially in a housing market where rental housing is always in short supply. Unfortunately the children and families are often the ones to suffer—either with a continuing hazard, eviction, or an untenable increase in rent.

In addition, we have no contractors who bill themselves as deleaders nor do we have a certification requirement—a situation that will need to be addressed in the near future.

The medical management of lead poisoned children has been more encouraging. Perhaps this success story is most visibly evidenced by the increase in numbers of screenings by private providers from 32 in 1984 to 3,500 screenings in 1987. Our success in enlisting the support of New Hampshire's family practitioners and pediatricians can be attributed to a process that began when the first positive cases were found in our Well Child Clinics, and has continued throughout the Program's operation. Whenever a positive screening or confirmatory venous is processed by the Public Health Lab, contact is made by telephone with the child's physician with recommendations for confirmatory testing on capillary screenings and an offering of the Program's medical protocols, the CDC's 1985 statement and consultation with one of the Program's pediatric consultants. In addition, the medical protocols have been mailed on two occasions to all pediatricians and family practitioners in the state.

Rounding out our Program is the educational component. In the initial two years of the program an educational brochure on lead poisoning, an educational cartoon about FEP and a poster warning of occupational/avocational exposure as a source of lead for children were developed. Since the Program's inception, a high profile has been promoted through regular articles in a newsletter which is sent to most physicians and health agencies in the state. Childhood Lead Poisoning Prevention Week has been observed with the cooperation of our Governor who has issued a proclamation each year.

This year's Lead Week had as its theme "Renovate With Care," urging that anyone doing renovations have their paint tested for lead and then observe appropriate precautions. With the assistance of True

Value Hardware Stores, we redesigned and printed our brochure and created a renovation poster. The brochures and posters were distributed to health and human service agencies, health officers and to the 70 New Hampshire True Value Hardware stores around the state for display and distribution during the week and thereafter.

Once again, utilizing much information from other states, and in the absence of adequate rules for our law, we've designed a booklet, *Guidelines for Safe Abatement*, which is provided to health officers, landlords, parents of lead poisoned children, contractors and anyone seeking information on safe paint removal.

We have also participated on the NECCLPP Educational Task Forces—as Amy has described to you; an investment which has benefitted every state.

Where do we go from here? There will always be room for improvement and development within the Program. But the progress within the last year is encouraging. An increased level of support by our Division of Public Health has been evidenced by a willingness to sponsor legislation this year requesting state funding for program support and development. Although the funding legislation was not successful, money from the Preventive Health Block Grant was made available to cover screening costs in our Well Child Clinics and to maintain our XRF's over the next two years. Our two major projects for this year are a revision of the rules for the Lead Law and refining our system of case management. Yes, we have far to go, but we couldn't have developed to this level without the investment and support of a regional network like NECCLPP.

AN ASSESSMENT OF LABORATORY ISSUES IN CHILDHOOD LEAD POISONING PREVENTION

Patrick J. Parsons, Ph.D.
Noel V. Stanton, M.S.

Introduction

The field of analytical chemistry is a constantly changing one. New and improved analytical methods appear frequently in the literature. Advances in modern technology have brought forth many new instruments. The laboratory aspects of lead poisoning prevention are no exception to these changes. However, no matter how advanced an analytical laboratory may be, the quality of its data depends upon the quality of the collection procedures employed.

Specimen Collection Procedures

The most common procedure used to collect blood specimens in screening for lead poisoning is the fingerstick method. However, some minor differences in tools used to puncture the finger may be found. Some collectors prefer to use a Microlance,™ a thin piece ofstainless steel with a sharpened point, claiming it results in a better "stick." Others prefer to use the Autolet,® an automatic lancet that consists of a spring-loaded needle and stabilizing platform that, allegedly helps reduce the trauma and injury associated with obtaining micro-blood specimens from very young children. Ultimately, the selection of either Microlance or Autolet is a matter of personal preference for the collector.

If the sole objective is to analyze for erythrocyte protoporphyrin (EP), then no special procedures, other than proper cleaning, are required prior to the "stick." Specimens of whole blood are usually drawn into microcapillary tubes containing sodium heparin, or another suitable anticoagulant, and mailed to the laboratory for analysis. In some cases, blood from the fingerstick wound is collected on filter paper for analysis. One advantage of this is the lower cost of filter paper compared to capillary tubes, and the ease with which specimens can be collected. The major disadvantage, however, is that no immediate follow-up analysis for blood lead is possible for elevated cases, and in addition, the data quality for EP obtained using dried blood spots on filter paper suffers from poor precision. This is caused by the variety of ways in which blood drops can be transferred to the filter paper in addition to inherent viscosity differences between blood specimens. However, this mode of screening provides more reliable

quantitative data for EP than that obtained with the hematofluorometer.

The determination of blood lead presents a different set of problems for collectors, one of which is contamination. Scrupulous cleaning procedures are necessary prior to collecting blood specimens. The finger should be cleaned first with soap and water, then with an alcohol swab and finally, a barrier film of silicone or collodion™ should be applied, using either a spray can or an impregnated swab. This last step ensures that the skin surface is isolated from the blood thus preventing contamination as well as helping the blood drop to bead. It is common for two microcapillary tubes to be used for each patient since an elevated EP can be followed up by analyzing the second tube for blood Pb. Needless to say, samples of *all* materials employed in the collection procedures, including swabs and Critoseal™ putty, should be checked for Pb contamination. Properly trained collectors, working together with competent laboratory staff, should be able to provide high-quality analytical data.

Determination of Blood Lead

Techniques for the routine determination of blood lead (Pb) fall largely into two categories: Atomic absorption spectrophotometry (AAS) and anodic stripping voltammetry (ASV). Both are element-specific techniques, that is, they are set up and calibrated for one particular element. Although it is possible to determine other elements, this is more practical with AAS rather than ASV.

Atomic Absorption Spectrophotometry can be further sub-divided into a number of instrumental methods which, although based upon AAS, are different. For many years, prior to development of micro-sampling accessories, Flame AAS was utilized by extracting Pb into methylisobutylketone and aspirating into the flame. This technique is not quite so popular today, because use of solvent extraction-AAS has been superseded by that of microanalytical attachments with the spectrophotometer, that not only greatly improve detection limits and sensitivities for many elements but, moreover, require much less sample volume (10-50 uL compared to 3-4 mL for Flame AAS). One of the first such attachments was the Delves cup, so named after its inventor, who found that inserting a small cup, containing an aliquot of dried whole blood, into an air-acetylene flame caused rapid vaporization of the blood matrix relative to Pb. This difference in vaporization rates could be utilized if a hollow silica tube was placed in the optical path. Examination of the resultant absorption profile reveals a distinct lead peak appearing after the "smoke" thereby enabling an absorption measurement to be made. This technique has been used frequently for many years for blood Pb although recent developments in electrothermal atomizers (ETA AAS), particularly graphite furnaces, have led to Furnace AAS becoming the technique of choice. Although routine blood lead determination by Delves cup AA is relatively straightfor-

ward and quick, it does not lend itself to automation, as does Furnace AAS. Modern Furnace AAS instruments now come equipped with automatic background correction and microprocessor-controlled calibration. Of course, a fully equipped Furnace AAS instrument with automatic sampling and injection accessories is quite expensive (*ca.* $40K-50K) especially when compared to ASV (*ca.* $11K).

Anodic Stripping Voltammetry is an electrochemical technique that is relatively simple but nevertheless does require a trained technician to operate the instrument. It is not particularly fast and there are some problems with incomplete decomplexation of the lead which gives rise to falsely low values. This is probably due to insufficient time allowed for full equilibration of lead with the Metexchange® Reagent.[1] With sufficient training and experience, accurate results are obtainable with this technique.

The minimum reportable concentration for Delves cup AAS is 1 ug/dL; for ETA AAS it is 0.1 ug/dL; for ASV it is 5 ug/dL.

Determination of Erythrocyte Protoporphyrin by Extraction

One toxic effect of lead is that it interferes with heme synthesis, resulting in elevation of protoporphyrin levels within the erythrocyte (RBC). Testing for elevated EP levels is widely used to screen for undue lead absorption. The traditional analytical method for the determination of EP involves a two-stage extraction procedure, first with an ethyl acetate-acetic acid solution, which removes porphyrins and heme from the cell debris, followed by extraction of "free" porphyrins from the organic phase into hydrochloric acid solution.[2] Conventional molecular fluorometry is utilized for quantitative analysis; calibration is normally carried out against protoporphyrin IX (PPIX) standards. In recent years, attempts have been made by several laboratories to agree upon a standard extraction method. EP is usually reported in concentration units of ug/dL whole blood, although some prefer to correct this to ug/g hemoglobin (Hgb), using individual Hgb values, or to ug/dL RBC using individual hematocrits. Although some other minor differences still remain, a consensus procedure has been developed, copies of which are available from either of the authors.

Although this consensus method has been generally accepted by many laboratories in the field, one outstanding problem remains. This is the true millimolar absorptivity for PPIX. The sole manufacturing source of PPIX, the primary calibration standard, is Porphyrin Products, Logan, UT. Because of the difficulties manufacturing vials with a known mass of PPIX, produced by hydrolysis of the dimethyl ester, the resultant stock solution needs to be standardized against the millimolar absorptivity using molecular absorption spectrophotometry.

The absorptivity, known formerly as the millimolar extinction coefficient, is a constant derived from Beer's Law that can be used to determine the concentration of a given compound in solution. For many years, the value for PPIX was assumed to be 241.[3] Indeed, much

of the clinical database covering EP levels is based upon this value. Recently, however, its validity has been challenged. It appears that the true value is nearer 297,[4,5] but a definitive value has yet to be published. Therefore, most laboratories have agreed to continue using 241 until a correct value is published *and* the CDC revises its risk classifications for EP in its booklet on Childhood Lead Poisoning Prevention.[6] Thus, it may be a year or two before this is fully realized. What is perhaps more important, however, is that *all* laboratories conducting tests for EP by extraction should adhere to a common method with an absorptivity value of 241, to avoid confusion until a definitive value is published.

Determination of Erythrocyte Protoporphyrin by Hematofluorometer

It has been shown that a large fraction of the protoporphyrin within the RBC is loosely bound to zinc. The labile zinc protoporphyrin (ZPP) complex has different fluorescence properties compared to "free" PPIX (FEP). Additionally, the ZPP/FEP ratio is not constant for all species; *e.g.*, in humans it is approximately 0.90. Therefore, attempts to equate ZPP with FEP number for number are erroneous.

The hematofluorometer was developed by researchers at the Bell Laboratories and was designed to be a portable screening instrument, set up in a physician's office or clinic, to test for iron deficiency or lead poisoning.[7] Briefly explained, the instrument operates on the principle of front-surface fluorometry. Whole blood specimens are placed on a glass coverslide that is inserted into the instrument. The resultant signal is derived from the fluorescence of zinc protoporphyrin, within the RBC, and its intensity is a function of the zinc protoporphyrin/hemoglobin ratio. The majority of hematofluorometers in the field, however, are calibrated to read in concentration units of ug "equivalent" erythrocyte protoporphyrin per 100mL whole blood, *i.e.*, they are calibrated to the extraction method. Of course, one has to assume an average hematocrit for a given population. Two options are generally available, 35% for a pediatric population, 42% for an adult population. Minor variations in real hematocrits introduce some errors into the reported value, as do minor variations in the ZPP/FEP ratio. Another source of error is the presence of fluorescent artifacts, in plasma, that increase the fluorescence signal.[8] Other factors that can result in false positive readings include elevated bilirubin levels[9,10] and drugs such as Depakene (Valproic acid).[11] Most hematofluorometers require blood to be oxygenated prior to making the measurement. This is due to the differences between oxyhemoglobin and deoxyhemoglobin absorbance characteristics. Incomplete oxygenation is a major source of error with hematofluorometers. The combination of these errors contributes to the unreliability of results obtained with hematofluorometers. They should not be viewed as substitutes for the precise, accurate determination of erythrocyte protoporphyrin by extraction. As a primary screening tool, however, they are adequate for identify-

ing children with elevated EP levels, false positives notwithstanding, provided they are properly calibrated against the extraction method. Unfortunately, the accuracy of values > 35 ug/dL cannot be trusted.[12]

The difficulty of achieving good calibration with hematofluorometers has been partly due to the lack of good reference materials. This situation has changed recently with the availability of several commercial control materials. The validity and stability of these standard reference materials has been the subject of an interlaboratory study, the results of which show that there are deficiencies with some of them.[13] This study has been conducted by the New York State Department of Health in cooperation with the Wisconsin State Laboratory of Hygiene and the Centers for Disease Control, and is funded by the Federal Division of Maternal and Child Health, Bureau of Health Care Delivery and Assistance, DHHS. Publications of these results is expected shortly as the study draws to a close.

Another recent development in the hematofluorometer field has been the introduction of a new instrument by Helena Laboratories, Beaumont, TX. A major novel feature of this instrument is that it is calibrated to report values in SI units; *i.e.*, umol ZPP/mol heme. Another novel feature is the use of a special reagent to treat the blood prior to taking a reading *in lieu* of oxygenating the specimen. The use of SI units is a significant departure from the conventional units of Aviv and ESA instruments, and of course, the extraction method. Since so little is known about these new units it is difficult to interpret them. Moreover, no absolute arithmetic conversion can be made between these and other units. Further work is required to find an empirical relationship between the two sets of units and, more appropriately, to correlate blood Pb levels with the ZPP/Heme molar ratio.

Conclusions

Advances continue to be made in the quality of data generated by laboratories conducting tests for Pb poisoning. Participation in external Quality Assurance programs is an essential part of good laboratory practice. In recent years, the Federal Division of Maternal and Child Health, Bureau of Health Care Delivery and Assistance, DHHS has funded a national QA program for erythrocyte protoporphyrin determination, in cooperation with the CDC and the Wisconsin State Laboratory of Hygiene. This program has been successful in helping laboratories around the country to improve their analytical skills.

Future developments include a national blood Pb QA program, operated by Wisconsin State Laboratory of Hygiene and evaluated by the CDC. This program, due to begin in mid-January 1988, will pick up the blood Pb program formerly operated by the CDC. In addition, a video film, funded by DMCH, has been produced by the Wisconsin Laboratory on the use of hematofluorometers, which will be made available to users of the instrument. In New York State, a proficiency

testing program has been established specifically for hematofluorometer users. The test materials, which are blood-based, are obtained from lead-dosed animals; the plasma is removed and replaced with a citrate-glycerol solution.[14] Target values are established by a consensus of reference laboratories using properly calibrated hematofluorometers. These reference materials are stable when refrigerated for approximately 16 weeks, and can therefore be used either to calibrate hematofluorometers or as quality control materials. They may be obtained through the New York State Department of Health's Wadsworth Center for Laboratories and Research.

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FINDING THE SOURCE OF LEAD

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Introduction

Several years ago I recall receiving a request from a physician at Children's Hospital of Michigan to investigate the environment of a three-year-old boy who was hospitalized and undergoing treatment for thallium poisoning. It was important that the source of thallium which caused this youngster to become poisoned was identified and removed. If not only to prevent his re-exposure to this highly toxic chemical upon release from the hospital, but also so as not to imperil anyone else.

How does one begin going about conducting the environmental epidemiology of a case of thallium poisoning? This was the opening question posed to lead investigators that attended Detroit's Environmental Management Training Program.* The correct answer, of course, focuses on the need to know of the many uses of thallium. Hence, knowledge of the uses of thallium, or any other similar toxicant under investigation, is the first order of business in trying to find it.

Therefore, finding the source of lead in the living environment of a lead-poisoned child hinges on the investigator's knowledge and understanding of the many uses of lead and its compounds, as well as the environmental pathways by which it enters the body.

Sources of Lead in the Living Environment

It is often said that lead is ubiquitous on our good ship planet Earth. This is so because lead has such multifarious uses. The extensive use of this very soft, bluish-white metal can be attributed to its durability, malleability, and its readiness to alloy with other metals such as tin and antimony. Moreover, its most valuable characteristic is its resistance to corrosion. This protective factor makes its use as a preservative ingredient in paint, in addition to its covering quality and desireable drying effect, both attractive and functional. Once its durability was recognized, the addition of lead compounds in paint formulation, especially red lead, resulted in it becoming standardly used throughout the world for protecting iron and steel against corrosion. This useage logically carried over to its inclusion in household paints

*The Detroit Health Department's Envronmental Management Training Program is supported by a grant from the U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, and is designed to train selected individuals in methods of lead hazard identification and abatement.

and for the same reasons—its durability and protection of exposed exterior surfaces. Unfortunately, it has been estimated that over 27 million dwelling units in the United States have been contaminated with lead-based paints. Over the years, as these housing structures fall into disrepair and the paint film deteriorates, the flaking, peeling, and cracking leaded paint becomes readily accessible to young children living therein. Moreover, even intact surfaces covered with leaded paint are a hazard to pica children.[1]

Dr. Julian J. Chisolm of Baltimore's Johns Hopkins University School of Medicine reported that the most common cause of lead poisoning in children is the ingestion of lead containing paint.[2]

In building construction, lead is used in ornamental lead-work on buildings, roofings, spouts, plumbing, and expansion bolts.

For industrial, production, and consumptive purposes, lead is used in the making of babbits, solders, cast and bonded pipe linings, lead hammers and anvils, electric cable coverings, and is an excellent alloy. Lead is also used in the manufacture of automobile batteries, in making lead sheets and coatings, and in making lead caulking. In the petroleum industry, the use of lead is not restricted to its addition to motor fuel for its anti-knock utility, but it is also used in the construction of corrosion-resistant equipment and in the oil refining process itself.[3]

Automobile manufacturers and auto collision shops use lead as a filling-in substance to cover scratches and hollows in body surfaces. Lead is used in printing, in the making of plates and print type. Its use in ammunition results in lead shot and the lead bullet. In musical instruments lead is used in the making of pipe organs and player pianos.

Domestically, lead is used in the making of crystal glass, ornamented jewelry, as a glaze on ceramic pottery and enamel, and in some rubber goods.

Lead compounds are used in certain pharmaceuticals and, earlier in our history, it was used in the treatment of stomach disorders. The protective quality of lead is not limited to the inhibition of corrosion. It is extensively used in medical clinics and hospitals to shield medical personnel from x-ray radiation. It is also used for the same purpose in the manufacture of rubber gloves and aprons. In the laboratory it has many uses; it is added to certain compounds in controlling acids and was used in pesticides. Lead even found its way into use in eye cosmetics by Asian families. One formula (Surma) was found to contain 88% lead sulphide.[4]

The Lead Industries Association reported in its 1984 annual review based on data from the U.S. Bureau of Mines that 1,490,072 short tons of lead were used as follows in alphabetical order (in tons):

ammunition—52,732
bearing metals—5,156
brass and bronze—7,667
cable covering—13,528
gasoline anti-knock additives—171,710
pipe, traps, and bends—15,058
sheet lead—16,168
storage batteries—954,291

casting metals—17,424
caulking lead—4,372
foil—9,133
terne metal—9,080
other unclassified uses—24,967[5]

The overall use of lead in 1984 (1,328,233 short tons) declined as compared to 1977 (1,490,072 short tons). Moreover, the use of lead in the manufacture of batteries and in gasoline as an anti-knock additive continues to dominate all other uses of lead in the United States.

As it has been shown, the uses of lead are many and essential to both our national economy and our quality of life. In the early twenties one author even entitled his book on lead: *Lead—The Precious Metal.*[6]

However, lead becomes a problem when it enters the human body by way of the alimentary and respiratory tracts in concentrations greater than the body can excrete. Overdoses of lead gradually build up in the body and cause lead poisoning. As William Ticky so succinctly put it in his book, *Poisons, Antidotes, and Anecdotes,* each small dose in itself may be handled as if it were harmless, but as with the straw that broke the camel's back, there's one dose that maims, cripples, or kills.[7]

Equipped with a working knowledge of the many uses of lead enhances the lead investigators' ability to determine the form of agent lead and the manner in which the child with undue lead became exposed to the agent.

Considering that lead-based paint is the highest dose source of lead and the most common cause of undue lead absorption in children, any environmental epidemiologic investigation for lead hazard should first focus on the condition of the painted surfaces inside and outside the child's dwelling. Unless, of course, another lead source has been positively identified as the causative agent.

When lead-based paint is not found to be the hazard, the investigator should look for other contributing lead sources such as: the content of lead in the house dust; the concentration of lead in soil; the concentration of lead in the atmosphere; in the water supply; improperly glazed ceramic pottery; printed materials; mop and broom handles; furniture (especially furniture painted over); other household items; toys; battery casings; and other heavily leaded objects found in and around the house, such as fishing sinkers, weights (draperies), keys, folk remedies or medicines such as Azarcon or Greta, Pay Loo Ah, Kohl or Surma, and other important medicines, ashes or fumes from wood burning stoves or fireplaces, ammunition, and lead wall-anchors. It is also important to consider the occupations and hobbies of the parents. Parents working in lead-related industries can bring home lead-rich dust on their work clothing, shoes, and hair.[8] In Detroit, we identified several children whose source of lead exposure was traced to the parents' occupation in a lead-related industry. Some parents were lead solder grinders working in auto plants. In such cases, it is important to determine whether such exposure was a single incident and isolated, or if other workers' children were similarly exposed but undetected. The investigator should make every effort to encourage

the parents of all the workers similarly employed to follow OSHA rules and regulations to protect themselves and their families and to bring their children to a clinic for a lead test. If indicated, a special lead screening effort should be scheduled to include the testing of all the children of lead workers, as well as the workers themselves.

Lead in the soil should be suspected as a possible source of lead ingestion for young children, especially the soil within three feet of the housing structure or other buildings around which children frequently play. One study conducted in Detroit by Ter Haar and Aronow showed that heavy concentration of lead may accumulate in the soil within a three-feet perimeter of the housing structure with painted exterior surfaces.[9] This is believed to be caused by prior scraping, flaking, peeling, or weathering of the exterior painted surfaces.

Other investigative considerations as sources of lead exposure are: the proximity of lead smelting plants, battery reclamation plants, automobile bump shops, and heavily trafficked streets to residential areas where young children live. However, the latter concern appears to be somewhat less important than initially believed. In a paper published by Ter Haar and Chadzynski, the lead level of children living within 200 feet of a heavily traveled street compared with those children living at a distance greater than 200 feet from a heavily trafficked area were not significantly higher.[10]

In summary, the lead investigator should keep in mind that the primary cause of childhood lead poisoning in the United States has been found to be lead-based paint and this should warrant the major focus of the epidemiologic investigation until it has been ruled out as the cause. However, this focus should be observed in view of the lowered lead level action trigger recommended for the environmental followup of children in Class II, whose lead levels may be attributed to other sources of exposure such as lead enriched dust or soil contamination.

Environmental Epidemiology

Case Review

When the investigator is notified of a case of undue lead absorption, the notification should trigger an orderly process. The orderly process begins with a review of the case. This is an intelligence gathering function that is essential to the subsequent field investigation. It's a child-centered approach, i.e., a child has been detected with undue lead absorption, it's a current condition: what caused it?

The referral report will usually indicate the severity of the child's condition which should determine the priority of the investigator's response. In this regard, it is important that the investigator know the risk classification of each child. (The Center for Disease Control statement entitled, *Preventing Lead Poisoning in Young Children*, is used to classify children into risk categories.)[11]

Other important information that is often available and should be sought out is the physician's findings and the nurse's report. For example, the physician's remark may indicate: "X-ray shows opaque material in the gut." The nurse's report may read: "Mother says child is pica for paint," or "Father works in lead-related industry." These are very important clues as to what the investigator should be looking for. Knowing a child has pica should result in a high index of suspicion on the part of the investigator to consider looking for substances that can be chewed and ingested. Pica is defined as a craving to ingest non-food substances. Many investigators of lead poisoning in children point to its close association with pica.[12]

Hence, in reviewing the referral report, the investigator should consider in addition to the foregoing the following: (1) the proficiency of the laboratory that does the analysis; (2) high dose versus low dose sources of lead; (3) the child's current medical status, i.e., in-patient or out-patient; and (4) the area in which the child's home is located.

Relative to the laboratory analysis, most laboratories that analyze blood for lead and erythrocyte protoporphryn participate in the proficiency testing program conducted by the Public Health Service, Center for Disease Control. The investigator should apprise himself of his laboratory's proficiency in analyzing these blood specimens. Nothing is more frustrating than to investigate the environment of a child classified as a Class IV child (urgent risk), find no lead hazards, and later learn that serial tests were found to be in the normal range and that the laboratory erred.

Based on knowing the child's risk category, i.e., a Class IV as compared to a Class II child, should be helpful to the investigator in looking for high dose sources of lead as the cause. Class IV cases might ingest or chew on lead sources such as leaded paint or lead-containing objects like toy lead soldiers, fish sinkers, etc., whereas the source of lead for a Class II child may be soil or dust. However, the investigator should not be misled by the risk category because the Class II child may have just started to get into high dose lead sources.

The child's current medical status is a consideration because if a child is an in-patient, more often than not, additional intelligence is available from the physician treating the child that is most helpful in finding the lead source causing the condition.

Knowing the area in which the child's dwelling is located is of major consequence in identifying lead hazards. Is the housing structure located in an older section of the city containing houses built prior to World War II, a time when lead-based paints were commonly applied to interior and exterior wall surfaces? Is there a lead smelting plant nearby or an automobile bump shop located next door to the child's dwelling? Is it in an urban renewal area where housing demolition is taking place? From the foregoing, the need for a comprehensive case review is quite obvious and should take place as the first fact-finding step of the orderly process used in finding the source of lead.

Epidemiology is that branch of medical science which investigates the causes of epidemics and determines approaches to control them. It deals with the study of diseases of groups rather than of individuals. The application of epidemiologic principles is helpful in narrowing down the range of environmental insults which cause disease. More often than not they serve to pinpoint the specific agent. In childhood lead poisoning control activities, an epidemiologic approach is used; however, on an individual case basis, it is the investigation process that leads to the identification of the lead hazards in the child's environment, and subsequently, to their abatement.

A major component of the environmental epidemiologic process is the case interview of those persons most familiar with the habits and characteristics of the child including the mother, father, guardian, siblings, other relatives, playmates, babysitter, and the like.

Case Interview

The second step in the orderly process is the case interview. The interview is of major focus and has been found to serve two important purposes. Primarily, it is most useful to the investigator extracting information on the whereabouts of lead hazards in the child's environment. Second, it serves as an educational forum for the respondent and results in a better understanding of the lead problem which allows them to contribute to its resolution.

The investigator conducting the interview must be an expert in his field and should be knowledgeable of lead and its uses. Furthermore, (s)he should understand all aspects of lead poisoning since (s)he is expected to provide information and answer questions knowingly. (S)he should know exactly what information (s)he needs and obtains it before the investigation. The following are some of the questions asked during the interview:

Does the child ingest, chew on, or put in his mouth painted articles like crib/bed, toys, guardrails, window sills, or fallen paint flakes?

Does the child pick at painted surfaces to get flakes or chips, putty around windows or soft metal objects?

Does he/she play with toys, jewelry, ammunition, beads, fishing sinkers, or any items with solder on them?

Does he/she drink or eat food that has been prepared, stored, and served in ceramic containers?

Does he/she put matches, cigarettes (including their ashes), cosmetics, dust, or soil in his/her mouth?

Where does the child sleep? Where does he play?

Where does he/she like to hide?

Where and how does he/she spend his waking hours?

Locations where the child spends his time?

Do you and/or your husband work? Where?

What are your occupations?

Who is your babysitter?

Where do you think your child may be getting his lead?

In addition to the demographic and environmental type questions asked in the interview, questions such as those presented above provide the investigator with clues as to where to begin the environmental search for lead.

Environmental Investigation

The third step in the orderly process is the environmental investigation. Upon completion of the interview, the investigator begins his/her room-by-room inspection of painted surfaces using an X-ray fluorescence lead detector. The XRF fluorescence lead analyzer is a portable instrument or device used for the identification and measurement of lead on painted surfaces by X-ray fluorescence analysis. The instrument contains a radioisotopic source that upon release stimulates the lead atoms in the paint. This stimulation causes the atoms to fluoresce and the instrument reads this fluorescence in milligrams of lead per centimeter squared (mg/cm^2) of paint surface tested.[13] Attention should be given to hallways, closets, pantries, foyers, porches, attics, basements, stairs, steps, guardrails, and enclosed porches, as the inspector moves from room to room.

The investigator records the readings of the XRF for each wall and woodwork surface tested by room. XRF readings should be randomly taken from at least two locations on each wall. Paint color is also noted, as is the presence of peeling or flaking paint on the walls (including ceilings) and any incidence of chewing and nibbling.

The interior inspection is followed by an exterior inspection and XRF readings are obtained from the porches, bannisters, guardrails, ballusters, roof support columns, exterior window sills, stairs, treads, and exterior wall surfaces including the wood trim of brick or masonry structures.

Outbuildings, including garages, sheds, barns, wooden fences, laundry posts, children's play equipment, picnic tables, outdoor furniture, including exterior buildings adjacent to the residence being inspected, and any other painted surfaces are measured for lead content. XRF readings are duly noted for each paint surface tested.

XRF readings of 0.7 mg/cm^2 should be considered positive. It is important to note that the lead analyzer is a probability sampling device and repeated readings are necessary for reliability.[14]

Soil should also be considered for testing especially that soil within a three-foot perimeter of the house or on a vacant lot on which a housing structure was razed, if the child is known to play there. Soil samples are obtained using a scoop or small hand shovel, placed in a glassine envelope, plastic bag or container (lead free), appropriately

labeled and submitted to the laboratory for analysis. The quantity of soil required varies between laboratories, the investigator should inquire of his chemist what amount he needs. The final step in the orderly process is abatement methods. Following the exterior inspection, the lead investigator should return to the premises and discuss the findings with the parent or guardian, recommend a course of immediate action to temporarily abate the identified lead hazards and apprise them of what must be done to permanently remedy the situation.

If lead hazards are not identified at the child's primary premises or there is reason to believe that there are lead hazards at other locations frequented by the child, the investigator should proceed to those locations and continue the investigation.

Other locations may include the homes of relatives and friends, the babysitter's house, day care centers, schools, and nursery schools, or a park or playground. The investigative procedures remain the same but a separate record should be completed for each location inspected and collated with the child's case records.

From the foregoing procedures one should conclude that the primary focus of the investigation is on lead-based paint. This is as it should be for in the over-whelming number of cases of children with lead poisoning the cause of the child's lead body burden has been attributed to lead-based paint. Cincinnati reported that with truly rare exceptions, the source of exposure for all cases of childhood plumbism has been interior and exterior household surfaces—walls, ceilings, window sills, porch railings, etc.—that have been covered with numerous coats of lead paint.[15] In Philadelphia, a physician from children's hospital writing in *Child Health* stated the single most important culprit of childhood lead poisoning in America remains the peeling of lead contaminated paint which was so widely used a quarter of a century ago.[16] In Milwaukee, more than 60 percent of the dwelling units inspected for lead hazards between 1972-1974 were found with lead paint hazards. Moreover, for the first five reporting quarters 89 percent of the dwellings inspected were found with lead hazards.[17] A three-year study in Chicago reported the significance of the housing structures in cases of lead poisoning. Slum areas with deterioration of buildings, walls, and furniture covered with repeated coats of lead containing paints and the resulting continued exposure produce optimum conditions for this disease.[18] The Cleveland study clearly showed the relationship between old housing and childhood lead intoxication. Twenty-seven percent of the children screened living in old housing were detected with abnormal lead urine levels compared to the less than three percent who lived in a newer housing project. Subsequently, five percent of the children from the old housing were diagnosed with plumbism, whereas no cases of plumbism were identified in those children living in the new housing project.[19] The same pattern held true in Detroit. Over the years, lead-based paint hazards have been found in more than 90 percent of the dwellings inspected.

However, if *no* lead paint hazards are found, the environmental search should continue until the lead source is found and includes the collection of samples of soil, dust, water, and household items for wet chemistry analysis. All suspect items should be analyzed for lead content. Dust and air-borne particulate compounds of lead may be removed from the air for chemical analysis using either high-volume or personal air samplers when atmospheric air is suspected.[20] The techniques and methods of collection to be used for soil, dust, water, and air samples relative to time, sample volume, type of containers, storage, etc., should be discussed with the chemist of the laboratory that will analyze the samples, as these vary between laboratories.

Detroit's lead investigators routinely carry with them in the field an environmental sampling kit for sampling paint, plaster, dust, soil, dirt, or other environmental samples and includes: (1) pocket knife, (2) small pen light, (3) glass and plastic containers with caps, (4) self-adhesive labels, (5) moist paper tissue wipes, (6) 200 square centimeter templates (for measuring wiping area), (7) small disposable brushes, (8) small trowel and scoop, (9) tablespoon, (10) plastic bags (with ties or self sealing), (11) long-handled scraper, (12) adhesive, Scotch, and masking tapes, and (13) tweezers. (All items are either lead-free or measured for lead content.)

Ideally, it would be most desirable for the investigator during a case investigation to obtain samples of dust, soil, water, paint, air, and any other household items so as to determine in total the level of a child's exposure to lead from all sources. However, the costs to conduct this type of investigation on each and every case are prohibitive. The investigator must then be selective in his choices of samples collected and the extent of his investigation in many cases is limited by the resources of his program or agency.

At the conclusion of the dwelling unit inspection, the investigator should evaluate all of his findings to determine if (s)he has found the most probable lead source(s) contributing to the child's added body burden. The objective of the investigation is to establish a cause-effect relationship. The evaluation should include the application of the fundamental epidemiological principles—agents, host, and environment—in terms of the relationship of time, place, and person.[21]

As it relates to time, childhood lead poisoning is a chronic condition which usually occurs over a period of time depending on the length of exposure, the amount of lead ingestion, and its absorption. Hence, relative to time, the investigator should consider what the child does during his waking hours, where he spends them, how, and doing what. The localization of time and the length of exposure is helpful in pinpointing the lead source. For example, how long the child has lived in his present dwelling is an important consideration. Perhaps the family just moved in and the child got his lead diet at a prior residence.

Plumbism usually results from the body's intake of lead over a

period of weeks or months of excessive amounts of lead. The amounts and frequency of lead ingestion can decrease or increase the time for symptoms to appear, if they appear at all.

Place relates to the child's total living environment and the sources of lead within it that are readily available and, of course, is linked to how the child uses his time. Two lead poisoned Detroit siblings were often reported to play in a partly finished, dimly lit attic of a neighbor's house whose owner's daughter also served as their babysitter. The investigator found one damaged wall, between the top and lower parts of a bunk bed, found to contain lead paint that the children were getting into just before they took their afternoon nap.

Host or person is the child with undue lead absorption. The investigator should consider his/her age, sex, habits, characteristics, ethnic groups, social, and economic attributes and the like. For example, knowing that a child has pica, is a dirt eater, sucks his thumbs, or is a nail biter may help identify the environmental pathway by which the child obtains lead. A lead investigator from the Louisville, Kentucky, Childhood Lead Poisoning Prevention Program learned that a child liked to suck or mouth things. After an exhaustive environmental search without any success in locating lead hazards, the investigator continued to probe for the lead source in a discussion with the child's parent. "Haven't you noticed anything unusual that the child has been getting into?" he asked. "Perhaps something that you took away from him?" The young parent left the room and returned with a handful of 22-long bullets and asked, "Do you think these may have caused the problem?" Upon observing the bullets, it was evident that pieces of the lead slug were missing and appeared chewed on. Eureka! The child had once been observed playing with the bullets taken from a drawer in the parents' bedroom. This case made for a simple abatement—the bullets were removed, permanently.

Lead poisoning should be evaluated in the foregoing terms and plausible causal relationships established. The causal agent (lead) is found broadly throughout the environment and in many forms. When it becomes readily available to an unwitting and susceptible child, it's only a matter of time for lead poisoning to occur in its usually insidious style.

Therefore, in conducting the environmental epidemiological investigation, the investigator should know the many uses and sources of lead, the many environmental pathways along which it travels, the mode of intake (ingestion or inhalation) and the person. We have found this orderly process to be a most effective and efficient method of finding the source of lead in the living environment of a child identified with an added body burden of lead.

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LEGAL ASPECTS OF CHILDHOOD LEAD POISONING PREVENTION

Edward L. Schoenbaechler, J.D.

An Outline of Substantive Law

Federal Jurisdiction

A. A good source for the background on federal action in the lead poisoning area can be found in the Federal Register, Vol. 49, No. 88, Friday, May 4, 1984, p. 19210. This contains the introductory comments of the Department of Housing and Urban Development (HUD) as part of the administrative rule-making process.

B. Generally, local public health departments have little contact with any federal law dealing with lead hazard abatement. There are principally two areas of federal concerns of which we should be aware:

   1. The Lead-Based Paint Poisoning Prevention Act of 1971, 42 U.S.C. §4821, et seq., which authorized agency action to prohibit the future use of lead-based paint in Federal or federally-assisted residential construction or rehabilitation, and to provide for the abatement, "as far as practicable," of the hazards of lead-based paint in existing housing covered by HUD mortgage insurance or housing assistance payments. The regulations themselves can be found in the Code of Federal Regulations (CFR) at 24 CFR Part 35.

   2. The Consumer Products Safety Commission also is concerned with lead levels in toys, cribs, and similar items of more general interest.

State Jurisdiction

A. General authority.

   1. Each state government is a sovereign entity with the inherent power to act in the name of the people of the state for their general good. No power of government is greater than the power to protect the public health and welfare through the exercise of the "police power."

      a. The power of the state is exercised by the state legislature through its elected representatives.

      b. Since the legislature lacks the time and the expertise to deal with the details of regulation in most areas, e.g., public health, the authority to act is delegated to a specialized state agency like the State Health Department.

Acting within the scope of its delegation and pursuant to specific statutory authority, the agency is given power to enact administrative regulations which have the force and effect of law. This rule-making power is the essence of administrative law, and is found at all levels of government—federal, state, and local.

B. Lead Poisoning Regulation.

1. Every state health department can be presumed to have the authority to regulate lead hazards. Since lead-based paint is acknowledged to be a public health problem, and since such agencies are already given the statutory mandate to abate public health nuisances, then no additional specific authorization is necessary to enable a state health agency to act in this area.
2. This action would likely take the form of agency proposal and adoption of lead poisoning regulations under its rule-making authority.
3. Of course, there is always the possibility of specific statutory enactments by the state legislature.

Caution: Unless carefully drafted, state legislation may be viewed as prohibiting local regulations which may be necessary, and which are usually more stringent than statewide requirements. A thorough analysis of this doctrine of preemption by legal counsel would be appropriate. For an example of one state's difficulty with state lead statutes, see Commonwealth v. Do, Inc., KY, 674 S.W.2d 519 (1984).

Local Jurisdiction

A. The comments above concerning the inherent power of state government to act to protect the health and welfare of the public apply as well to local government. Whether it be a city or county, the statutory authority and common law power of the elected, governing body, is sufficiently broad in scope to permit the regulation of lead poisoning. This type of regulation usually takes the form of a local ordinance or resolution.

B. In addition, local health departments may have the authority to adopt rules and regulations pertaining to matters of public health and the abatement of nuisances. If so, this should surely extend into the area of lead poisoning prevention.

C. As suggested above, care should be taken in proposing such ordinances and regulations so as to avoid the possibility of state preemption problems. The case of Commonwealth v. Do, Inc., *supra*, gives excellent support to the position that local lead regulations are valid as supplemental to state interests.

An Outline of Procedural Law

Order of Abatement

A. The first step toward securing compliance with the health code is to locate the violation, then issue to the responsible party an order to abate the nuisance.

B. The order should be clear enough on its face that the citizen knows what is expected of him.

C. *Always Try to Deal Person to Person.*

1. We cannot expect the public to act responsibly to an order if we do not take the time to help them understand the reason for the order.
2. Simple due process requires that a person have adequate notice of governmental action affecting them. If you have never dealt with the citizen directly, the prosecutor may dismiss your case in court on the theory that the citizen was denied a hearing.
3. The court process should not be seen as the answer to all of your difficult cases! Many things can go wrong on the way to the courthouse. You are well advised to make every effort to handle the problem in the field, one-on-one, where the possibility of successful abatement is much greater.
4. Similarly, the order for abatement should routinely include an invitation for an office hearing, along with a telephone number to call to request such a hearing.

The Administrative Hearing

A. Use of the administrative hearing process, also called the office hearing, is somewhat time consuming but affords sufficient benefit that it must be encouraged.

1. It forces the field sanitarian to operate on a person-to-person level, which invariably produces better results than notices left on the door or placed in the mail.
2. It satisfies any insistence of the prosecutor that the defendant be afforded a hearing prior to the institution of criminal enforcement proceedings.
3. It enables the sanitarian to *listen*, and thus to gather information which may otherwise be unavailable or difficult to obtain.
   a. If the order of abatement is directed at an individual who does not own the property in issue, you would be much better finding that out at this point, rather than while standing before the judge. Your credibility with the judge and prosecutor is most important and must be carefully guarded.

b. In some cases we need additional information, such as when the owner has died and we are unable to locate all of the heirs in order to secure compliance. Often the person to whom you send the order will be most willing to give you the names and addresses of the others responsible.

Consent to Search

A. Inside a dwelling unit.

1. As a general rule, the person with possessory interest in the dwelling unit has authority to allow the sanitarian on the premises to conduct a routine inspection.
   a. The tenant does have authority to let you inside their own dwelling unit without the need for the landlord's consent.
   b. Any adult member of the household will do; it would not be proper to rely on the consent of a minor.
   c. Adult babysitters pose something of a special circumstance, and common sense will help identify those situations when it would be advisable to return another time.

B. Outside the dwelling unit.

1. Generally, you have a right to be where other members of the public have a right to be. Thus, if the mailman, newspaper boy and UPS driver all open the gate and go to the door, there is no reason for you not to do the same.
2. Walking to the rear of the property may or may not be advisable, depending on your purpose.
   a. If you are only looking for the owner so you can speak with her, it is probably all right to check the back of the house.
   b. If this is your first inspection, in response to a complaint, it may be appropriate to make a brief inspection in the back to determine whether there is cause for the complaint and need for follow-up investigation.
   c. If this visit is to determine if a hazard has been abated, and therefore may result in court action, it is probably best to avoid the technical trespass and either come back or get the information another way.

C. If you cannot get consent to search and have been told to stay off the property, stay off the property! There are, however, alternatives.

1. Remember that any information you gather in violation of the law will be inadmissible in court.
2. Interview the complainant and neighbors; their testimony can be very valuable if they are willing to come to court.

3. As long as you are where you have a right to be, the information you gather (or things you see) is admissible. Thus, if the neighbor will permit, go next door and look over the back fence. Use binoculars to view property from a longer distance if necessary.
4. Get a search warrant.

The Search Warrant

A. The process for getting a search warrant is greatly simplified if you can present the prosecutor or department counsel with the necessary facts. Attached to this outline as Appendix A is an Application for Search Warrant that has proven helpful in providing the needed information.
   1. Be sure to obtain all of the information requested by the Application, for it can avoid some embarrassing and costly mistakes, such as serving the warrant on the wrong house!
   2. If the information you have is from a child protective service worker or other governmental employee, note that on the Application, including the name of the individual; such information is credible and will be convincing to the judge who must approve the warrant.
   3. Do not forget to take along the police and the animal control officers if such difficulties are likely to be encountered.

B. Use careful discretion before seeking a search warrant. The time of the judge and prosecutor is valuable and should not be wasted on the marginal case, or the case with no significant health risk.

The Criminal Complaint

A. If all else fails, you may need to start criminal enforcement proceedings. (We assume that your agency does not have the authority to conduct trials and assess administrative fines without court action.)

B. Understand that this is a criminal proceeding. As such, the court must get personal jurisdiction over the defendant. To do this, the defendant must be personally served with the criminal complaint by a peace officer.
   1. This means that in the case of absentee landlords, you will not be able to get service of process and the case will be dismissed.
   2. Corporations, limited partnerships, and other legal entities also own property and can be criminal defendants. Who to serve, and how to get them served, are technical questions that can only be answered by legal counsel based upon the law of your jurisdiction.
   3. Remember that you must rely on someone else to serve the warrant. If their efforts are ineffective, you lose.

4. The moral of the story is: Don't expect the court system to be the answer to all of your problems. Like any machine with many moving parts, it breaks. If you can secure compliance in the field without the need to resort to court action, your success rate will be much improved.

C. The purpose of the criminal process is still to secure compliance and elimination of the nuisance. This is often done through the use of a substantial fine, suspended on the condition that the nuisance will be removed and that the property will remain in compliance with the health laws for a period of time, usually two years.

Trial Issues

A. If you finally get the defendant in court, and he won't plead guilty, you will probably have a trial. You will need to offer proof to the court that the defendant is guilty as charged.

B. What is "proof"?

1. Generally, proof is any information which will assist the finder of fact in its deliberation.

   a. Photographs of the scene, with paint flaking on the ground, are excellent examples of proof.

      • Note that you do not need to be the person who took the photograph in order for it to be admissible; you only need to be able to testify that it "fairly and accurately reflects the conditions that existed" at the time of the event.

   b. A simple narrative of what you observed is proof that those events did in fact occur.

   c. Physical samples of paint chips taken from the scene are highly persuasive, but to be admissible you must establish the "chain of custody." (See below)

   d. Along with the paint samples is the testimony of the laboratory expert who analyzed the sample and can testify to its lead content. Establishing the chain of custody is a prerequisite to the admission of this testimony.

   e. Particularly persuasive is the result of a field analysis using an instrument similar to the XK-3™ x-ray fluorescent analyzer.

      • In order to be admissible, the person operating the unit must be able to testify that: (1) the instrument was working properly at the time of the field test; and (2) the person knew how to operate the instrument properly.

      • This is particularly good testimony because: (1) judges and juries all like "show and tell"; (2) people tend to believe anything the high technology instruments tell them; and (3) the machine is not subject to cross-examination.

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- Note also that it is very helpful if the instrument has a read-out that corresponds to the legal standards directly, without requiring a conversion to a different unit of measurement.

2. What we are concerned about here is not just the admissibility of testimony, but the persuasiveness of the testimony as well. Thus, we may choose to use more than one of the examples described above in presenting our case.

C. The "Chain of Custody"

1. In order to admit into evidence any physical item, e.g., paint samples, you must assure the court that the integrity of the evidence has been secured.
2. This means that you must be able to identify each person who had physical possession of the evidence, and they must be available to testify that they did not alter or tamper with the evidence while it was in their possession.
3. This also means that the evidence must be kept in a secure location, such as a locked file cabinet, so that the possibility of anyone else tampering with it is reduced.
4. If you can show the full chain of custody, then the chemist will be able to testify to the results of his lab analysis on the sample itself. Without the chain established, the test results will be inadmissible and you may not be able to prove illegal lead content.
5. If you have any questions or need help in establishing proper chain of custody techniques, just ask your local narcotics officers for their assistance. They have special evidence envelopes which simplify the process and help secure full information from all personnel.

APPENDIX A

LOUISVILLE AND JEFFERSON COUNTY DEPARTMENT OF PUBLIC HEALTH

APPLICATION FOR SEARCH WARRANT

Property Owner: _____

Address: _____

City: _____

Blk: _____ Lot: _____

Deed Book: _____ Page: _____

Description of Location: (cross streets; residential; sewers, etc:

_____

_____

Description of Premises/Building: _____

_____

_____

★ ★ ★ ★ ★ ★ ★ ★

Nature of the Health Violation: _____

_____

_____

Sanitary Code Provision Violated: _____

Facts Observed by Health Dept. Worker (Please identify persons and dates): _____

_____

_____

_____

Facts Observed by Witness (Please identify, include addresses of all persons and dates): _____

_____

_____

_____

Will witness sign Affidavit in Court? _____

Will witness testify? _____

★ ★ ★ ★ ★ ★ ★ ★

Area Sought to Inspect: _____

_____

Who Will Inspect: _____

Will Police Assistance be Needed: _____

* * * * * * * *

Sanitarian: _____

Division: _____

Supervisor: _____

Telephone: (ext.) _____ When to call: _____

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LEAD AS A MEXICAN FOLK REMEDY: IMPLICATIONS FOR THE UNITED STATES

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Abstract

Use of lead to treat the folk illness, "empacho," is common in Mexican culture. Data collected in Guadalajara hospitals indicate that 34-36% of the populations treating empacho used such remedies. Lead based remedies are also known in other areas of Mexico. These data suggest that in situations of lead poisoning among migrant populations in the United States of Mexican origin, use of folk remedies should be investigated as a possible cause.

Introduction

This paper discusses the availability and use of toxic folk remedies in Mexico and suggests the relevance with regard to health particularly among migrant populations in the United States of Mexican origin. These data represent the first two stages of a longer term project designed to investigate the extent to which lead oxides are used in the treatment of gastrointestinal problems in Mexico. Ultimately the goal of the project is the development of an education program to discourage use of remedies of this type.

Background

In the early 1980's, it was discovered that lead oxides were being used as a folk remedy among Mexican-American populations in the United States (Sankury et al. 1983, Ackerman et al. 1981, 1982, Vashistha et al. 1981, Bose et al. 1983, Trotter 1985). Both greta (lead oxide) and azarcon (lead tetroxide) were used to treat "empacho," a folk illness, which is believed to be caused by something being stuck in the digestive tract, resulting in diarrhea, and/or vomiting. The samples of lead oxides collected from users in the United States had been largely purchased in Mexico. Beyond this, not a great deal was known about the situation in that country. It was clear that greta and azarcon were in use in some areas for the treatment of empacho. An acute case of lead poisoniong due to treatment of empacho with azarcon was reported in Mexico City (Montoya Cabrera et al. 1984). The

two year old patient had been treated several times a day with the amount of azarcon that could be picked up with three fingers. This azarcon had been purchased near Tolucca; other members of the family had also purchased the substance in Acapulco (Montoya Cabrera et al. 1984). Both azarcon and greta were reported in use for treatment of empacho in Guadalajara and nearby villages (Garcia et al. 1986). In addition, there have been several poisonings and at least one death in the Guadalajara area caused by the use of greta for empacho (Cueto: personal communication).

Methodology

Goals of the first year (1986) included assessing the extent to which azarcon, greta, and any other lead based remedies were available in Mexico, and determining where geographically and through what kinds of vendors these remedies were sold. Twelve sites throughout Mexico were selected for investigation. The criteria used for selection were:

1. Major center of distribution of herbs and other folk remedies through the market system (Mexico City, Monterrey, San Luis Potosi, Guadalajara, Oaxaca);
2. Areas where greta and azarcon were reported to have been known or used (Guadalajara, Mexico City, Tzintzuntzan, San Cristobal Las Casas, Tuxtla Guiterrez);
3. Areas where low fired pottery, employing the use of lead oxides in the glazes, was made (Patzcuaro, Tonala, Tzintzuntzan, Puebla);
4. Areas where a large percentage of the population were of an Indian cultural background so that issues of cultural variation of patterns of use of lead oxides could be explored (Tzintzuntzan, Oaxaca, San Cristobal Las Casas, Merida).

The methodology used was to visit the herb stands in the markets, and inquire of the vendors as to remedies for empacho. If greta and azarcon were not mentioned, the vendors were specifically asked about them, how they were used, and where they could be purchased. The drug, hardware, and other stores they suggested were visited, and attempts were made to purchase these substances in those locations, as well as to gather information from clerks and customers as to knowledge and use of lead based folk remedies. In addition, in as many areas as possible, drug wholesalers were visited, and the remedies under investigation were purchased, if available.

The second stage of the project (1987) was designed to investigate the extent to which lead based remedies were chosen by mothers as remedies when their children had gastrointestinal complaints. The availability of lead oxides in Guadalajara (found in 1986) lead to that city being selected for further investigation. This paper reports on the

preliminary results of interviews in two hospitals in Guadalajara, the Hospital del IMSS No. 46, and the Hospital Civil. Five days were spent interviewing in each hospital. All women who entered either the Pediatric Outpatient, or Pediatric Emergency waiting rooms between the hours of eight in the morning and two in the afternoon were questioned initially by Mexican social workers to assess whether they recognized and treated empacho. Those women who had treated empacho in their own children were asked an additional series of mostly open ended questions dealing with the topics of causes, symptoms, and treatments of empacho, specific use of lead based remedies, and sources of lead in the environment of the house or neighborhood (such as use of lead glazed ceramics, or nearby battery factories or busy streets). The interview was administered as the women were waiting for their children to be seen by medical personnel.

Results

Distribution Networks

Initial investigations indicated somewhat different distribution systems for greta and azarcon. The center of greta production in Mexico is the industrial city of Monterrey. Greta, as well as azarcon, has extensive industrial uses, in producing ceramics, rubber, and plastics. But greta is generally seen by the public as a glaze for pottery, and is primarily distributed through hardware stores which specialize in products such as paint and cement (tlalaperias). The greta is put on after the ceramic vessels are painted to make them shiny, and protect them from flaking. The vessels are then fired again, though at relatively low temperatures. In centers of pottery production greta is available at the level of the local store (tienda). As a pottery glaze it is perceived to be completely legal. However, it is reported to be used as a means for causing an abortion ("They say it can be used for that," suggested a male pottery maker in Tonala), so women who try to purchase small quantities of it (for example, only a few hundred grams), are viewed with extreme suspicion, as abortions are officially illegal in Mexico.

Azarcon is distributed for industrial uses; in addition it is available through networks of drug stores and drug wholesalers, as it is necessary for making an externally used dermatological preparation called, "agua de vegeto." Both drug stores and drug wholesalers sell at the retail level. Mexican drug stores are of two types, farmacias, which sell pre-packaged remedies, and boticas, which will actually mix up a prescription, such as "agua de vegeto." The former are much more common. Azarcon is usually sold by drug wholesalers and drug stores of the botica type. It also occasionally appears outside of drug sales outlets at the level of the market stand. It seems to be considered legal by the drug wholesalers, though ostensibly only for external use. Only in an occasional botica and market was sale of azarcon for internal use perceived to be legal.

Networks of distribution of both greta and azarcon cross. For example, in San Luis Potosi, a market vendor referred a request for greta to a hardware store. The opposite was observed in Merida, where, when seeking greta for ceramic uses in a hardware store, a referral was made to a drug wholesaler. A request for azarcon in a drug store in San Luis Potosi was referred to a hardware store. Thus, networks of distribution of greta and azarcon as treatments for empacho, and in their industrial uses are not entirely separate. Vendor referrals suggest that the substances can be acquired from a variety of different kinds of sources, and that their customers have no conceptual problem in being sent to look for a gastrointestinal remedy in a hardware store, or a pottery glaze at a drug wholesaler.

Purchase of Lead Based Remedies

Purchase of greta was easiest in areas where low fired pottery was made. Locations where greta was purchased (Table 1) included Tzintzuntzan, where the substance was purchased in a tienda (small store). In Tonala, greta was being sold in a person's home. In the latter location, the greta was taken from a bag of 25 kilos which identified the name of the distributor in Monterrey, as well as the chemical composition of the substance—lead oxide (oxido de plomo). Laboratory analysis confirmed that both greta samples were high in lead content (Table 1).

The purchase of large quantities of azarcon was very simple. At a large drug wholesaler in Mexico City, one and a half kilos of azarcon were purchased. While it was sold in the context of other drugs, it came labeled as a poison. Many of the clientele in this store were from other parts of Mexico, and used the opportunity to purchase drugs not only for themselves, but also to sell at home. However, it is not necessary to come to Mexico City to make purchases through these large wholesalers; one can call in an order, and it will be sent to you. So the availability of azarcon through drug wholesalers in the capital essentially means that it is available to anyone, anywhere, who seeks to purchase it.

As azarcon is recognized by the Mexican medical community to have legitimate external medicinal applications (Hurtado: personal communication), the drug wholesalers who distribute azarcon are acting completely properly, and indicate their awareness of the toxic nature of the substance through the poison label which is put on packages of azarcon. However, in Mexico, it is common for substances of all types to be purchased in bulk, and then broken down into smaller sized units for sale to the consumer. This is apparently also the case with respect to azarcon. It was purchased at one botica, and at one market stand, and in both cases the samples were not labeled as to the contents, or their poisonous nature. Laboratory analyses of all three azarcon samples confirmed their high lead content (Table 1).

TABLE 1
Chemical Analysis of Samples

Sample	Lead Content
1. Azarcon (Mexico City)	95.0%
2. Azarcon (Guadalajara)	93.3%
3. Azarcon (Oaxaca)	93.3%
4. Greta (Tonala)	94.1%
5. Greta (Tzintzuntzan)	97.3%

Data suggested that another lead based remedy might also be in use in Mexico. On several occasions, in discussions of the use of greta and azarcon for the treatment of empacho, another substance, albayalde, was mentioned. Therefore, it seems likely that lead carbonate ("albayalde" in Spanish [Hawley 1975]), a white powder whose most common use is to make paint, may be in oral use for the treatment of empacho. Ingestion of this substance poses the same potential health problems as do greta and azarcon; in the process of digestion, lead carbonate is converted into lead oxide (greta).

Use of Lead in the Treatment of Empacho

Interview data collected in the 1987 field season confirmed the patterns observed the previous year, and indicated widespread use of greta, azarcon, and albayalde on the part of consumers (Table 2). The situation is even more serious than would initially appear to be the

TABLE 2
Use of Lead in the Treatment of Empacho

Location	Hospital No. 46 del IMSS Number	%	Hospital Civil Number	%
Women not treating empacho with lead	65	66%	56	64%
Women treating empacho with lead	33	34%	31	36%
Total population treating empacho	98	36%	87	33%
Women initially questioned	270		261	
Population at risk	33	12%	31	12%

case, in that a number of the mothers often used lead to treat several of their children. The number of people treated by the 64 mothers who use lead to treat empacho is at least 94, as in several cases, the women said they had treated all of their children, without specifying the number. Thus, about 12% of the households of the populations sampled in each hospital can be considered at risk from the use of these substances. The most common treatment for empacho, reported by 48% of the women, is a three part treatment consisting of massage, a spoonful of oil, followed by an herbal tea. Use of lead is the second most common treatment, reported by 35% of the mothers, and differs from the above treatment only by the addition of a pinch of lead to the oil ("the amount you can pick up with three fingers"). This pattern contrasts with the situation in the United States where lead is particularly used in the treatment of severe empacho (Trotter 1985). Thus, lead is a first line approach to the treatment of empacho in Mexico, as opposed to a last resort in the United States.

The Guadalajara mothers' concern for their children who they believe to have empacho may be related to the seriousness with which they view this illness. Forty percent of them felt that a child might die if he/she was not cured of empacho. Severe cases of empacho are most commonly treated by taking the child to a doctor or hospital, but the women reported that they were frequently laughed at or scolded by doctors if they explained that their children had empacho. Yet the label "empacho" covers symptoms which Western health care workers also feel are worthy of medical attention. Physicians who examined the children considered by their mothers to have empacho most frequently diagnosed the children as having enteritis or gastroenteritis, or a combination of gastroenteritis and other problems (Table 3).

The children given lead were so treated between 1956 and 1987, when they were between one month and 5 years of age. They were given doses of azarcon, greta, or albayalde ranging from a pinch to 3 teaspoons, and were treated between one and five times each, with a mean of 2 treatments per child. Unlike the situation in the United States where lead is a home remedy (Trotter 1985), in Mexico the lead is frequently administered by a curandera (native healer). Thirty-seven percent of the children who were treated with lead received it when their mothers took them to a curandera, while in the other cases, the mother obtained the lead herself or from a friend, relative, or neighbor (Table 4).

Conclusions

These data have a number of implications for the situation in the United States. For example, in the studies done of patterns of use of greta and azarcon in the Southwest of the United States (Trotter 1985), regional differences in use were discovered. Greta was preferred in Texas, while use of azarcon was more common in Arizona. Based on

TABLE 3
Physicians Diagnoses of Children Considered by their Mothers to Have Empacho

Diagnosis	Number	Percentage
Enteritis or gastroenteritis	11	37%
Gastroenteritis plus: infection; infection and dehydration; dehydration and malnutrition; food poisoning; infection, dehydration, malnutrition, anemia and dermatitis	5	17%
Prolonged diarrhetic syndrome	2	7%
Parasites	2	7%
Dermatitis	2	7%
Other	8	27%
TOTAL	30	

TABLE 4
Mothers' Sources of Lead Based Remedies

Source	Number	Percentage
Curandera	25	37%
Pharmacy, store, or herb store	29	43%
Friend, relative, or neighbor	6	9%
Another city	4	6%
Didn't remember	4	6%
TOTAL	68	

Note: Four women reported use of more than one type of lead oxide.

the findings in Mexico, it can be suggested that these patterns reflect not regional trends, but rather the origins of the populations involved. Mexican Americans who use greta may have migrated to the United States from areas of Mexico where low fired pottery is made, especially the areas around Guadalajara. They have probably traditionally obtained greta through ceramics or hardware store networks. Mexican Americans using azarcon are likely to have come from other regions of

Mexico, and/or are accustomed to using drug networks to acquire the substance.

Usage patterns in Mexico suggest that among the migrant population in the United States of Mexican origin, many of the mothers will have had no direct experience in treating their children with lead, it having been administered in Mexico by a curandera. They may remember the treatment, but may be unclear on the appropriate dose. This type of situation may result in some of the more serious cases of poisoning seen in the United States. The lack of familiarity with dose may also be why, in contrast to the pattern in Mexico, in the United States, other remedies are chosen before greta or azarcon (Trotter 1985).

While importation of greta and azarcon into the United States for internal pharmaceutical uses has been officially prohibited, it is clear that the United States is incapable of stopping the flow of people into the country from Mexico, much less that of their possessions. A small amount of lead oxide will serve for repeated doses of a child, so migrants entering the United States can easily carry a supply with them. Further, the rate of migration from Mexico to the United States shows few signs of diminishing greatly, and the area around Guadalajara is an important sending area of such migrants. What this means, then, is that the problem of use of greta, azarcon, and albayalde is not likely to disappear any time soon.

Further, networks of distribution of lead oxides as treatments for empacho, and in their industrial uses are not entirely separate. In addition, azarcon has legitimate distribution through drug wholesalers. Therefore, control of use of lead based substances as internal remedies for empacho through control of their sale is impractical, suggesting a focus on education as a way of approaching this problem. But the data suggest additional levels of complexity to be dealt with in educational programs which attempt to discourage use of such substances. Mexican mothers and health care professionals both label the symptoms associated with empacho, but failure of the health care workers to respect the mothers' diagnoses of empacho has led the mothers to avoid initially bringing cases of empacho to the attention of those trained in Western medicine. However, the assertion of interest in and respect for the mothers' perceptions on the part of the research team, even in the setting of a hospital, encouraged them to discuss these issues with us. We suggest that in addition to the types of consumer education previously undertaken in the United States (i.e., a poster compaign stressing the dangers of use of greta and azarcon), attention also be focused on educational efforts among health care workers who come into contact with Mexican American populations. If the mothers remain convinced that they will be scolded or laughed at by bringing children with empacho to the attention of health care workers, they will continue to use the remedies with which they are familiar. And they are quite familiar with lead oxides.

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FUTURE DIRECTIONS

J. Routt Reigart, M.D.

My charge today is to summarize what has gone on in this conference, give you a feeling for where we are, where we've been, and transmit a few of my ideas about where I think we ought to go in the future in dealing with childhood lead poisoning. As an introduction, I would like to go back 14 years ago to a conference that Bob Goyer organized on low level lead toxicity under the auspices of the National Institutes of Environmental Health Sciences. This conference occurred at the time when I was just beginning to work in lead poisoning. It had a great impact on my knowledge and understanding of childhood lead poisoning. Dr. Emil Pfitzer did a summary for that conference and as I was reading over his talk, in order to figure out what I should say today, I found a lot of what he said is still very relevant to us. Let me share some of his thoughts with you. Dr. Pfitzer started by saying,* "Alice had come through the looking glass and had just read an intriguing poem in a book she had found on the table. When she had finished it she said, 'Somehow it seems to fill my head with ideas only I don't exactly know what they are.' Many times in the past I have shared Alice's dilemma. Fortunately for most of us here, this is not our first conference on the toxicity of lead and it becomes increasingly less difficult to place pertinent ideas and data into perspective. We have really come a long way in our understanding of low level lead toxicity. How many conferences, symposia, books, review and research articles have there been in the eight years since the Public Health Service Conference on environmental lead contamination in December of 1965? Our numbers and efforts have expanded daily and data has been generated almost too rapidly for any one of us to keep up to date at all.

"As stimulating as this has been for the concerned and involved scientist, the past years have not been completely joyous ones; rapid expansion can bring its problems. For some, it has been a frustration of keeping patience while new investigators rediscover old facts. For some, it has been the frustration of watching repetitive research dollars being spent for months and years while new investigators learn new skills already available in established laboratories. And for some, it has been the frustration of feeling that their voices fall on deaf ears with no apparent impact on changes in environmental contamination. Despite these frustrations, who can deny that the end results have brought important new techniques and useful new approaches to long-standing problems?

*Pfitzer, Emil A.: *Environmental Health Perspectives:* Exp. Issue 7:247-252:1974.

"These advances in knowledge have been taking place concurrently with many other changes. Many of us grew up at a time when the final arbiter about health and disease was the physician. It was simply not respectful to question his judgment about what was good or bad for health. If any do not realize how far the pendulum has swung away from this tradition, let him speak with those who have recently sought federal funds for human experimentation. I, for one, have accepted the condition that statesmen and legislators will often be the decision-makers about factors that influence my health and yours. This is not an easy pill for the health scientists to swallow." As Dr. Pfitzer goes on, he says something else important to all of us today: "Many of us wear at least two hats in our daily activities. That of the involved scientist, and that of the involved citizen. Some of us also wear the hat of the regulatory administrator. Sometimes we are accused of wearing all our hats at the same time and forgetting which one is on top."

Dr. Pfitzer finishes with this very interesting conclusion stated fourteen years ago: "In conclusion, I find that the evidence is very suggestive that the most significant, subtle, and sensitive changes during low level lead exposure may be behavoior disorders in children rather than changes in heme synthesis. The methodologies to establish this relationship in a quantitative manner seem to be within our grasp, both from studies in humans and laboratory animals. The preponderance of evidence also identifies lead in paint, plaster, and dust in older housing as a major source related to current health problems, altough substantial efforts are being made to identify and control other sources."

Well, Dr. Pfitzer said 90 percent of what I need to say today. In 1973 when this conference was held we were just beginning to develop the science to define the risk of low level lead exposure to children. At that time we felt that we could cure lead poisoning quite easily. All we needed to do was get children's blood lead below 40 ug/dl. We knew how to treat them, follow them, and successfully reduce the blood lead to the "safe" level of less than 40 ug/dl. We could clean up the houses so that we felt it was safe to send children back to their old home.

Today, as we were clearly reminded during this meeting, things are not so simple. It is clear that there is no "safe" level of lead exposure. All of us are suffering at one level or another from lead poisoning. Treatment is not the easy thing we once thought it was and it is not easy to reduce lead exposure. Furthermore, it is an exceptionally difficult task to correct demonstrated hazards in our environment.

Let's go on to talk a little bit more specifically about what happened in this meeting. Overall the thing that strikes me the most is that our knowledge of the adverse effects of lead exposure has really gotten way ahead of our practical ability to deal with the problem of lead exposure. We now know clearly how hazardous lead is at even very low levels of exposure.

I have organized the talks into two basic groups, those that de-

fined effects of lead and those that discussed ways to deal with the lead. Dr. Lin-Fu gave us a very elegant discussion primarily related to where we have been. She reviewed very carefully the history of our past efforts in dealing with childhood lead poisoning. She encouraged us by reviewing some of the improvements that have occurred with active screening and intervention and by activities of the EPA in reducing lead in fuels. She discussed many of the myths that still exist about childhood lead poisoning and told us how to dispel them. She stressed very clearly the need to integrate the activities of all levels of providers in all levels of government and the private sector, and she pointed out that we have come back to our concerns about lead based paint and its hazards as we have had some success in dealing with other lead sources.

Dr. Needleman then spoke to us about what we have learned about lead neurotoxicity. I think probably the most important thing he said was "lead poisoning is a man-made disease." We sometimes forget that and we sometimes forget that since we made the disease, it is up to us to fix it. He reviewed many of his elegant studies on the effects of low level lead exposure, stressing lead as a neurotoxin. He stressed lead effects on I.Q. and lead as a major cause of maladaptive classroom behaviors. He showed us that in one population he had studied, the attributable risk of lead to the need for special classroom education was about 43 percent. This is an astounding figure! Dr. Needleman also reviewed the metanalyses which have shown that, despite some of the inconsistencies and conflicting conclusions between various studies of low level lead toxicity on intelligence, there is no question that lead exposure at low levels is damaging to the intelligence of children. He reviewed briefly, also, the effects of lead on stature and learning, and pointed out that lead appears to be a teratogen. He pointed out that the threshold for these effects is in the blood lead range of 10 to 15 mg/dl. He reminded us that greater than 50 percent of our black population are in this range, as well as a high proportion of our white children.

Dr. Bellinger, I have to give him the gold star. Dr. Bellinger's presentation for me was by far the most elegant talk of the week. Dr. Bellinger pointed out the importance of recent longitudinal studies and showed very clearly how they defined some very important issues, including the critical periods for exposure to lead, that is, at what point in development injuries of various sorts occur. He told us how these studies can help us better understand the critical dose at those critical periods. He further showed how these studies are very helpful in eliminating concerns about reverse causation which is the concept that the neurologic damage caused the lead poisoning rather than that the lead poisoning caused the injury. He showed us his very distressing data on the adverse effects of lead by fetal exposure in an educated, relatively affluent population at levels very slightly above population means. I think that as we progress one of the things that may really

help us is this broader understanding that it's the whole population who is suffering from lead, not just people who are poor or otherwise disadvantaged.

As he talked about his studies, he did give a glimmer of hope despite the distressing news that so many children were being damaged *in utero*. He pointed out that some of these effects may be reversible after birth by a nurturing environment with positive infant stimulation. This possibility of reversibility gives us some hope that active programs in infant stimulation may be useful as an immediate intervention during the inevitably long period that will be necessary to remove lead hazards from our environment. Dr. Bellinger also reviewed other longitudinal studies and showed how concordant they were with his observations.

Dr. Joel Schwartz from the EPA then reviewed some of the successes of efforts by the EPA in reducing lead in fuels and foods and showed us what a positive impact that has had on the overall lead burden of our population. He quickly turned around and stressed how difficult it may be to deal with some of the other lead hazards in our environment and in this gave a prelude to some of the later discussions. He showed us the distressing data on the effects of lead on birth weight and on the stature and weight of older children and then related this to studies of vitamin D activation and suppression of TSH. It's always powerful to observe the manner in which epidemiologic observations can be affirmed by basic laboratory science as in this case. He reviewed some very interesting non-behavioral neurological effects of lead including the statistical analysis that he's done which shows that at blood lead levels between 5 and 25 mg/dl significant hearing losses can be demonstrated. He also discussed some data which indicated that peripheral nerve conduction can be altered at levels much lower than many of us had thought in the past.

To this point in the discussion we had been presented with some very compelling data which fulfill the prediction of Dr. Pfitzer 14 years ago that we would be able to demonstrate that children are damaged at very low levels of lead exposure. The data is so compelling and it's so advanced that it's clearly far ahead of our ability to deal with the hazard. I think then it is time to look at what we heard in the conference about our ability to deal with the lead hazard that is so severe and ubiquitous.

I don't mean to demean the efforts of Dr. Graef or any of the physicians from the American Academy of Pediatrics (AAP) but the statement the AAP published and Dr. Graef reviewed shows that the AAP has abandoned what could be a very important leadership role in dealing with this enormous childhood program. I have always thought of the AAP as being a pro-active group that is concerned about the welfare of children and promotes the welfare of children. As Dr. Graef reviewed the statement of the AAP it is very clear that there was some excellent background material in it. I think it was important that the

AAP saw fit to publish this background material in a clear readable fashion in a journal which pediatricians read so that information that they may have ignored in the past is more accessible. As a statement of the AAP perhaps it will be more widely read than other publications. I am very distressed at seeing what happened when it came time to make recommendations. My interpretation of what happened is that the AAP said, "It is not our problem, it is the government's problem." Rather than coming out for universal screening, it told pediatricians that if they made a prudent decision to not screen a child they did not have to. It recommended screening of children only once or twice in their first 6 years of life which is far less than other persons or groups recommend for screening. What the AAP in effect told pediatricians is, "This is an important problem but you don't have to be a leader and you don't really have to worry about it unless you are in a 'high risk environment.'"

The AAP statement then made a series of recommendations for government action with which I cannot disagree. These actions would require a high level of governmental commitment and public expense but are very worthwhile. The problem I have is that this statement does not support and endorse a similar level of commitment by members of the American Academy of Pediatrics. The impression we are left with is that the AAP is saying, "It's not our problem, it's the government's problem, let the government handle it." Frankly, I find this to be an appalling position for a group that should be at the forefront of child health advocacy.

Dr. Julian Chisolm is the dean of workers in this area in our generation. He always seems to be one step ahead of the rest of us. In his talk, he emphasized the return to the concern about the old sources of lead in the environment. He talked about how we had gotten lead out of gasoline fuels and in so doing, decreased airborne exposure. He pointed out to us that all the "good" things that we thought we were doing back in 1973 involving the environment and medical intervention may in fact have either done little good or even worsened some problems. We really need to look at what we are doing in every area of management and I think that includes abatement of environmental hazards. Dr. Chisolm showed very clearly the contribution of dust to lead exposure and showed us how futile much of prior abatement efforts have been.

We heard from Ronnie Levin about the EPA effort. I think the EPA is an agency that is very concerned with doing something about lead exposure. What came clearly through from her talk was that the EPA has moved rather well, although sometimes in fits and starts, in dealing with some of the very important sources of lead which are relatively easy to deal with, such as lead in water supplies and lead in auto fuels. However, as she discussed removal of other sources of lead from the environment, it became clear we had a problem. We don't know how to do it. We don't have the methodology. The two areas she

spoke most of were the enormous contamination of our soils that has happened over many years and the disposal of wastes which come from incinerators, from houses being abated, from all of the sources of lead that must be disposed of or recycled. I think what came through clearly is that the EPA has a great deal of concern about the problems. However, in many of these areas adequate research isn't available and there are many practical barriers to solving the problems. It appears that we must do a great deal more in the future in devising appropriate and effective ways of dealing with the environment.

Carolyn Newton talked about the HUD regulations. I found these very discouraging and basically incomprehensible in light of current knowledge. I don't understand why HUD claims to not have the information available to write appropriate regulations. As I pointed out, Dr. Pfitzer said back in 1973 that dust was probably one of the major sources of lead and yet we heard that HUD didn't even know about dust as a hazard when the regulations were written. I suspect that there is fault on both sides there. I think that we as concerned health care workers have not been forceful enough in making such agencies aware of what the hazards are and what we consider to be appropriate intervention. I suspect that there is also some resistance within HUD to acceptance of that information as it represents a very difficult practical problem to that agency.

There are three particular areas in the regulation that are most distressing. First, these regulations still use children as the indicator of a hazard, only examing houses wherein children have already been found to be badly lead poisoned. Second, these regulations allow and encourage incomplete and inefficient abatement of houses by allowing limited correction of hazards such as removing paint from only one wall of a room if that wall is the only one that is not intact. It seems to me that if we're going to look at housing we ought to look at the whole house and all of the hazards in it because an intact wall today may not be intact tomorrow. It certainly is very short-term economy to fix only one wall and then have to go back a month later, six months later, and do the other walls. The third appalling aspect of the regulation is that it does not require relocation of children while the abatement is going on except when it is "judged" hazardous to the child. I know of no abatement procedures that are not hazardous to the child. It is clear that these regulations are at least 10 years outdated as written. Perhaps that is how long it takes to get them approved, but if the government can't do well with their own housing, I don't have any idea what the rest of us can do with the other 30 million houses in this country that aren't governmental. It seems to me that HUD should be taking the lead, rather than dragging their heels and staying many years in the past.

Let me conclude with some of my personal thoughts about the problem of lead poisoning today. It seems to me that as we are allocating our resources to move forward in the future, our research

funding ought to be directed in the following areas: First, we really need to look at new, unique, and innovative approaches to protect children from lead exposure on a primary basis. For the entire history of management of childhood lead poisoning we have practiced secondary prevention wherein we wait until children are already lead poisoned to intervene. I think it is time, as Dr. Chisolm and Dr. Graef said, to move forward in protecting children before they are lead poisoned. I think in attempting to protect children we shouldn't necessarily focus just on the lead. We ought to improve children's nutrition and implement other positive health interventions that help protect from lead hazards.

Second, as is very clear from Dr. Bellinger's talk, we need to learn how to protect our fetuses. I think that it is clear that that's a real and imminent danger and none of us at this point have any idea how to protect the fetus from intrauterine exposure and injury other than by reducing maternal lead levels.

Third, we need to understand much more about the chemical, biochemical, physiologic and pathologic aspects of lead damage. Further, we need to learn more about how, if possible, to reverse the effect of lead. The advances in these areas have lagged very far behind our advances in neurophysiologic and neurobehavioral testing.

And fourth, I think we need to spend a lot of money in figuring out how we are going to get lead out of the environment properly without increasing hazards to our population. I think that includes, as Dr. Chisolm mentioned, improved methods of abating houses, improved methods of disposing of the wastes, and consideration of methods of recycling the wastes so that we don't just continue to dump tons of lead contaminated material back into the environment.

Going from research, I think the next thing that I feel strongly about is that we must insist that our governmental agencies, particularly HUD, cease and desist in the governmental promotion of lead poisoning. I think all of us have just got to insist on that and exert a great deal of pressure on our governmental agencies. HUD ought to be taking the lead rather than following.

Next I think that all of us who are involved in dealing with children have to change a bit in our perception of what we are doing in our lead screening programs. For a very long time, and again this is one of the points well made by Dr. Lin-Fu, we have said that *any* lead exposure is toxic. However, in looking at our screening information, we have spent a great deal of wasted effort in attempting to separate "normal" from "not normal" children. We get into all sorts of discussions about whether we are getting all the "not normal" children by doing EP or ZPP's or this or that other form of screening. We have to accept that there are *no normal children.* And since there are no normal children, the major reason for screening, and I think screening should continue at a very high level, is to attempt to stratify children into greater and lesser levels of exposure and risk and injury so that we

have a better idea how to direct our limited resources. We must really accept and behave and work as if all children are lead poisoned. We must believe that what we are trying to do is take whatever resources we can get and use them in the best possible fashion. Screening by blood lead testing or EP testing is merely a way to decide where to put our money first. It is *not an attempt* to say who is "normal" and who is "not normal." When we begin to view things in this context we have far less problem with the tests and we are far more effective in our interventions.

Finally, I think it is clear that all of us must understand at this point the enormous ecologic disaster that we brought among ourselves. What we have done to our environment with lead pales compared to many other toxins that our population worries about. Everyone worries about dioxins and PCB's and possible radiation leaks from nuclear sites. Clearly all these things are real concerns which have attracted enormous public attention. For some reason, we have not done as good a job of getting the public to understand the degree to which all of us are suffering from this enormous pollution of the environment by lead.

As a final point, it is clear that we need a coalition of a wide variety of interested persons to work together to solve this problem. This coalition should represent state and local screening programs, physicians, laboratory personnel, nursing, environmentalists, nutritionists, lawyers and legislators at every level. Dr. Pfitzer said in his talk that if we can get our elected representatives to realize the enormity of this disaster and work to help us deal with it, we are going to be far more successful. And then the public: parents, communities, the private sector. Everyone who is involved in this problem, which is all of us, needs to work together toward common goals.

When we look at what an enormous problem lead contamination is, there is almost a desire to wring our hands and say there is nothing we can do about it. I think that is absolutely the wrong approach. It seems to be clear at this point that we've done an enormously good job of demonstrating scientifically to ourselves that *all* of us are damaged by lead. Now we've got to go on from there and let the rest of the world know that all of us are damaged by lead. We need to activate all of our agencies in research and development to try to deal with the problem.

I think this was a very exciting event. We have to thank Dr. Lin-Fu and the organizers. It is certainly the best lead poisoning conference I have been to in many years. Our needs and mandates for the future are clear. Let us get on with the task.

CLOSING REMARKS

Sarah Wilding, R.N., B.S.

The Kentucky Department for Health Services and the Louisville-Jefferson County Department of Health have received grant funding for 6 years from the federal Division of Maternal and Child Health to provide training in childhood lead poisoning management. The Project goals are to assist MCH staff in developing and/or increasing their efficiency in lead program management and to update individuals involved with lead program activities on new developments and activities of other programs. The major project activity is the provision of three 3½ day workshops in which technical information relating to the essential components of a childhood lead poisoning prevention program is presented. The workshops also provide a general knowledge base for the development and management of lead poisoning prevention programs, assessment of lead poisoning as a problem in specific areas, integration of lead screening with basic child health services and recognition of undue lead absorption as a public health problem and a Maternal and Child Health issue.

The second major activity of the Lead Training Project is the development of a semi-annual publication entitled *Lead Lines*. Articles and/or information of interest such as abstracts of published articles, research projects and educational offerings on childhood lead poisoning issues are included.

All registered attendees to this conference will be placed on the mailing list to receive announcements for the regular lead training workshops. You are invited to apply yourself or to pass the announcements along to a colleague. You will also receive a copy of *Lead Lines*. The next issue should be out in February-March. You still have time to submit articles to Mike Meyer if you act quickly.

Over the past 5 years the need for a national, multidisciplinary conference was developed in response to that need. Conference proceedings will be mailed to all registered attendees. We hope that additional copies will be available through NCEMCH and the Clearinghouse.

On behalf of the planning committee I hope that you have found the conference to be interesting and welcome your comments and suggestions for future offerings.

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